INSTITUTIONAL MICROBIAL ANALYSIS OF ODONTOGENIC INFECTIONS AND THEIR EMPIRICAL ANTIBIOTIC SENSITIVITY

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CERTIFICATE

This is to certify that this dissertation titled "INSTITUTIONAL MICROBIAL ANALYSIS OF ODONTOGENIC INFECTIONS AND THEIR EMPIRICAL ANTIBIOTIC SENSITIVITY" *is a bonafide work done under my guidance by* DR. ANEESH SEBASTIAN *during his postgraduate study period between 2010-2013 under* THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI, *in partial fulfillment for the award of the degree of* MASTER OF DENTAL SURGERY IN BRANCH III- ORAL AND MAXILLOFACIAL SURGERY *It has not been submitted (partial or full) for the award of any other degree or diploma.*

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CONTENTS

SPSS	-	Statistical Package for Social and Sciences
SV	-	Streptococcus Viridans
SA	-	Staphylococcus Aureus
PA	-	Pseudomonas Aeruginosa
EC	-	Escherichia Coli
KP	-	Klebsiella Pneumonia
CNS	-	Coagulase Negative Staphylococcus
PP	-	Peptostreptococcus
BT	-	Bacteroides,
AM	-	Actinomyces
SV+PP	-	Streptococcus Viridians with Peptostreptococcus
SA+PP	-	Staphylococcus Aureus with Peptostreptococcus
SV+BT	-	Streptococcus Viridans with Bacteroides
SA+BT	-	Staphylococcus Aureus with Bacteroides
CNS+BT	-	Coagulase Negative Staphylococcus with Bacteroides
SV+AM	-	Streptococcus Viridans with Actinomyces
SA+AM	-	Staphylococcus Aureus with Actinomyces

Abstract

INSTITUTIONAL MICROBIAL ANALYSIS OF ODONTOGENIC INFECTIONS AND THEIR EMPIRICAL ANTIBIOTIC SENSITIVITY

Introduction

Most purulent orofacial infections are of odontogenic origin. Odontogenic infections are the sequel to dental caries, periodontitis *etc.* Odontogenic infection ranges from periapical abscesses to superficial space and deep neck infection. In addition to systemic toxicity it also causes advanced complications such as suppurative mediastinal spread, an airway obstruction, retropharyngeal pleuropulmonary suppuration, haematogenous dissemination to heart valves, prosthetic devices, other metastatic foci, jugular vein thrombosis, mediastinal involvement, pericarditis, pneumonia, emphysema, arterial erosion, meningitis, and extracranial or intracranial extension of infection.

These infections continue to be a very significant cause of morbidity and mortality, hence early diagnosis and institution of immediate aggressive therapy is often essential. Hence knowledge of potential spectrum of pathogens as well as the antimicrobial susceptibility and regional resistance status is important for rational medicinal regime. Empiric antibiotics were administered before the culture and sensitivity tests results are available and specific antibiotics are selected based on the culture and sensitivity test results. Cultural analysis still remains the backbone of the clinical practice and the findings of a number of the prospective and the retrospective studies given a valuable insight into the bacteria which are often present. Regular study

of microbiological flora of abscess should be necessary to monitor adaptations and changes in the bacterial resistant strains.

Aims & Objectives

The aim of the study was to qualitatively evaluate different aerobic and anaerobic microbiota with their antibiotic sensitivity in head and neck space infections of odontogenic origin. To understand the efficacy of currently used empirical antibiotics in the management of odontogenic infections.

Methodology

All the Patients with head and neck fascial space infections of odontogenic origin who had not taken antibiotics were selected for the study. The site was anesthetized by local anesthetic (2% lignocaine with adrenalin). Pus sample were collected by transport cotton swab stick directly from the site for aerobic culture and also aspirated from the site and transferred to the Brain heart infusion broth transport medium for anaerobic culture. And it was sent immediately for aerobic and anaerobic culture. The culture was done in the Nutrient agar medium, Blood agar medium and MacConkey agar medium. The gram staining reaction is used to identify the pathogens in specimens and cultures by their Gram reaction and morphology. After material is dispended to culture media and incubated for 24 to 48 hours will visualize colonies of grown organism. The anaerobic incubation was done in an anaerobic jar. Then all sets of plates will apply biochemical tests to identify the genus and species of bacteria. Then the grown colonies of organism are spread over the Mueller Hinton Agar media plate. Labelled antibiotic discs are placed over the grown colonies of organism on the Mueller Hinton Agar media plate by the help

of sterilized forceps. This plate will be again incubated for 12 hours to 24 hours at 37^{o}C. A zone of inhibition is appeared surrounding the antibiotic disc indicates the sensitivity of the organism to the particular antibiotic and the zone of inhibition is measured by the help of WHO quality control Chart to access the sensitivity of above mentioned discs.

Results

In this study out of 142 patients, 125 cases organisms were isolated and taken up for the study, Out of 125 cases, 29 (23.2%) Aerobic organisms, 36 (28.8%) anaerobic organisms and 60 (48%) mixed organisms were isolated. *Streptococcus viridans* and was the most common aerobic organism isolated followed by Staphylococcus *aureus. Peptostreptococcus* and was the most common anaerobic organism isolated followed by *Bacteroides. Streptococcus viridians with Peptostreptococcus* was the most common mixed organism isolated followed by *Staphylococcus aureus with Peptostreptococcus.* Amoxicillin was the most commonly used empirical drug in all cases and showed highest resistance for all the aerobic anaerobic and mixed organisms. Among the Macrolide group, organisms were least sensitive to Erythromycin and higher resistant. Among the board spectrum antibiotics, organisms showed highest sensitivity and least resistance to Doxycyclin. Among the miscellaneous group, Linezolid was sensitive to all the aerobic, anaerobic and mixed group of organisms. Metronidazole showed sensitive to the entire anaerobic group. Clindamycin showed sensitive to the entire aerobic group. Organisms showed highest sensitivity towards Ofloxacin and Levofloxacin. Cefixime and Cefotaxime were showed highest Sensitivity among cephalosporins group.

Summary and Conclusion

According to the study there should be substitution of miscellaneous group of antibiotics such as Linezolid, or Clindamycin, third generation cephalosporins such as cefixime, cefotaxime, and fluroqunolones such as Ofloxacin and Levofloxacin for Amoxicillin in the empirical management of deep fascial space infections. Hence successful management of head and neck fascial space infections of odontogenic origin can be achieved by appropriate surgical intervention to establish drainage ,good overall supportive care of the patient, gram staining of purulent exudates to provide immediate information needed for the rational selection of an antibiotic and *in vitro* microbiological culture and antibiotic susceptibility.

Specificity of empirical antibiotic therapy could be improved with good knowledge about the pathologic flora in the locality. This will help in administration of appropriate antibiotics instantaneously to control the infection. This will counter the delay in identification of the causative agent and specific antibiotic therapy.

It can be concluded that the knowledge about the pathologic flora involved in head and neck infection in a locality and their sensitivity and resistance to commonly used antibiotics will help the clinician in administering appropriate antibiotics at the earliest phase of infection, which will adequately control the infection and hence minimizes the morbidity.

Introduction

Historically, the potential for a dental abscess to spread causing severe sepsis and death has been known since antiquity, although the role of bacteria in this process was not recognized until the turn of 20th century[1]. Head and neck space infections are known to be commonly of odontogenic origin. Earlier in the start of century neck spaces thought to be arise from tonsillitis, post operative tonsil wound, tuberculoid tonsil, tuberculoid cervical gland, caries of cervical vertebrae and syphilis of throat. These factors were considered as the immediate predisposing causes. Modern literatures point toward the deep neck infections arising from odontogenic infections. By the turn of 20th century, dental infections were associated with mortality rate of 10-40%[1]. When the Bills of Mortality began listing the causes of death in the early 1600s, 'teeth' were listed as the 5th or 6th leading cause for death[1]

Most purulent orofacial infections are of odontogenic origin[2]. Odontogenic infections are the sequel to dental caries, periodontitis *etc.* Odontogenic infection ranges from periapical abscesses to superficial space and deep neck infection. Deep neck and mediastinal abscess are rare but serious complication of odontogenic infections. But the spread of odontogenic infection accounts for 57% deep neck abscesses[1]. In addition to systemic toxicity it also causes advanced complications such as suppurative mediastinal spread, an airway obstruction, retropharyngeal pleuropulmonary suppuration, haematogenous dissemination to heart valves, prosthetic devices, other metastatic foci, jugular vein thrombosis, mediastinal involvement, pericarditis, pneumonia, emphysema, arterial erosion, meningitis, and extracranial or intracranial extension of infection[3,4], Carotid artery erosion is a devastating complication that can involve the common, internal, or external carotid branches. Internal jugular vein thrombosis is the most

common vascular complication. It is characterized by shaking chills, spiking fevers, tenderness, and swelling at the angle of the mandible or along the sternocleidomastoid muscle[5]. Internal jugular vein thrombosis may produce bacteremia, circulating septic thrombi with distant infection, or pulmonary embolism[5, 6, 7]. These complications clearly indicate the potentially serious nature of orofacial infections.

These infections continue to be a very significant cause of morbidity and mortality, hence early diagnosis and institution of immediate aggressive therapy is often essential. Knowledge about the potential spectrum of pathogens as well as the antimicrobial susceptibility and regional resistance status is important for rational medicinal regime.

It is well established that odontogenic infections are not caused by a single organism; instead these infections are polymicrobial in nature[1, 8, 9, 10, 11, 12, 13, 14, 15, 16] . These infections consists of various facultative anaerobes, such as the *Streptococci Viridans* group, the *Streptococcus Anginosus* group, and strict anaerobes, especially anaerobic cocci, such as *Peptostreptococci, Prevotella, Fusobacterium* species and *Bacteroides*[1]. Most of these infections were identified as mixed infections predominated by anaerobes of both cultivable and uncultivable origin. The flora associated with such infections is very much complex and it generally reflects the combined influence of the indigenous oral flora and the unique flora of the underlying condition[8].

Therapeutic strategies must be targeted at the degree of infection and host immune status. Some of them would require aggressive management, where as others require less aggressive modalities. The criterion for choosing an appropriate antibiotic or

its parent group is largely dependent on the type of organism involved and its virulence factor. Formulations in terms of specific culture guided therapy must be preceded by empirical therapy to limit the spread of infection.

Empiric antibiotics were administered before the culture and sensitivity tests results are available and specific antibiotics are selected based on the culture and sensitivity test results. Allergic or toxic reaction, swelling, temperature, and white blood cell count to decline after at least 48 hours of intravenous administration of pencillin, and a post operative CT scan shows inadequate surgical drainage are the reported reasons for therapeutic failure of pencillin. These findings suggest a correlation between infection severity and pencillin resistance and are the basis for the recommendation of clindamycin as the empiric antibiotic of choice in odontogenic infections[17].

Resistance may be either inherent or can be acquired by the processes of genetic mutation or gene transfer[18]. The mechanisms of acquired resistance fall into one of the five categories, although bacteria may employ more than one mechanism (i). Enzymatic modification or destruction of the antibiotic, (ii). Reduced antibiotic uptake into the bacterium (iii). Increased efflux of antibiotic from the bacterium. (iv). Alteration or production of a new target site (v). Over expression of the drug target[17]. Literature reports that the molecular biology of the antibiotic resistance by any one of the four ways. (1). Alteration of the drugs target site (2) Inability of the drug to reach its target. (3) Inactivation of the antimicrobial agent. (4) Active elimination of the antibiotic from the cell[17,18].

"Bacteria are cleverer than men" as they have the capacity to adapt in every environmental niche on this planet and now adjusting to a world laced with the antibiotics[19]. To combat the penicillin resistance, the synthetic antibiotics were synthesized, however resistance has also developed to these newer synthetic drugs[19]. The treatment of odontogenic infection is based upon three fundamental elements that are recognition of airway compromise, surgical intervention, and the administration of the appropriate antibiotic[20]. In most of the cases the antibiotics given are empirical and based upon the particular clinical condition of the patient. It may lead to inadequate treatment and development of bacterial resistance and multiple resistances.

Identifying the pathogen may be determined scientifically in the laboratory where the organisms can be isolated from tissue, pus, or blood. Antibiotic therapy is then either initial or definitive, and depending upon whether the organism is even identified precisely. Cultural analysis still remains the backbone of the clinical practice and the findings of a number of the prospective and the retrospective studies given a valuable insight into the bacteria which are often present[1]. Efforts made to identify the causative pathogens involved in the development of dental abscess in the past have been hampered by the inappropriate methods of sampling.

Microorganisms involved in infections can reproduce very rapidly and microbes such as bacteria can also freely exchange genes by conjugation, transformation and transduction between widely divergent species[17, 18]. This horizontal gene transfer coupled with high mutation rate and may alter by means of genetic variation which allows micro organisms to swiftly evolve (via natural selection) to survive in new

environment. This rapid evolution is important in medical field and has lead to recent development of super bug- pathogenic bacteria that are resistant to modern antibiotics. And also antibiotics were used indiscriminately and were prescribed even in mild infections and mild injury cases without knowing the chemotherapeutic susceptibility of microbes, this led to a challenging problem and development of more virulent strains of microorganisms which were more infectious and were resistant to many antibiotics, hence much more difficult to treat.

Therefore regular study of microbiological flora of abscess should be necessary to monitor these adaptations and changes in the bacterial resistant strains. More over on reviewing the literature it was found that lesser studies have been conducted on Indian population to list the common pathogens causing orofacial infection in our region. In India most of the patients suffering from orofacial infection are of low socio economic status with poor oral hygiene, undernourished or malnourished having below average autoimmune resistance and are deprived of proper oral hygiene. Another point is that without proper medical advice patients do take medication on their own as the antibiotics are also made available easily without appropriate licensed medical practitioner's prescriptions, which may contribute to the said problem. Under circumstances it becomes need of time to explore the resistance developed mechanism and its allied concerned problem so as to decide right medicinal therapy. Considering above facts this study has been designed to obtain valuable information of the laboratory data regarding the microbiology and antibiotic susceptibility of microorganisms causing head and neck space infections of odontogenic origin in our population.

Aims & Objectives

AIM

The aim of the study was to qualitatively evaluate different aerobic and anaerobic microbiota with their antibiotic sensitivity in head and neck space infections of odontogenic origin. To understand the efficacy of currently used empirical antibiotics in the management of odontogenic infections.

OBJECTIVES

The objectives of the present study were:

1. To identify the causative aerobic and anaerobic micro- organisms responsible for head and neck fascial spaces infections.
2. To evaluate the resistance against empirical antibiotics used in the treatment of head and neck space infections.

Review of Literature

Phillip H. Mann in 1960 conducted a study of identification and differentiation of culture of 26 coagulase positive strains of *Staphylococcus Aureus* and simplified by bacteriophage typing from a two hundred carious teeth from 200 patients. He has reported both the bacteriophage patterns and the susceptibility to penicillin, oxytetracycline, and oleandomycin (matromycin), Sixteen of the 26 coagulase-positive strains were non-typable or resistant to lysis by all the phages. Of the 25 coagulase-positive strains, 18 were susceptible to oxytetracycline, 25 to penicillin, and all to oleandomycin.

D. E. Hunt et al in 1970 conducted an in vitro study of antibiotic sensitivity of oral micro organisms to Actinobolin using a paper disk-agar diffusion method for inhibitory activity against cariogenic streptococcal strains, mixed microbial cultures, bacteria, and yeasts. The presence of zones of growth inhibition around the disks used as the parameter for the Actinobolin antimicrobial activity, and found that Actinobolin has in vitro antimicrobial activity against 9 cariogenic streptococci and 8 mixed cultures and a limited spectrum of gram negative and positive bacteria and yeasts, The organisms were highly sensitive to inhibition by actinobolin, and suggests that this antibiotic may be useful as a cariostatic agent.

Charles B. Sabiston et al in 1974[37] reported a research project to identify the types of bacteria present in abscess and their antibiotic sensitivity patterns. He found that *Fusobacteriem nucleatum* present in 7 of the 8 abscesses, *Bacteriodes* were present 4 out of 8 cases and *Facultative Streptococci* were isolated from 6 of 8 cases. Species of *Peptostreptococcus, Lactobacillus, Actinomyces* and gram negative facultative rods occurred in 2 cases. *S. Epidermidis* were found in 1 case. But the patients were not

responding to the pencillin therapy. Clindamycin was useful in treating the infections caused by pencillin resistant strains of *Bacteriodes* and anaerobic infection.

J. E. Turner, et al in 1975[33] conducted a study and determined the prevalence of bacteria associated with soft-tissue abscesses secondary to dental caries in 66 patients using aerobic and anaerobic culture and in vitro antibiotic susceptibility testing. *Streptococcus viridans* was the predominate organism and isolated from 42 (70 %) of the viable exudates, including 36 (%) in pure culture. *Streptococcus viridans* was susceptible to all antibiotics used in dental practice, except tetracycline. 8 (19 %) isolates of *Streptococcus viridns* were resistant to tetracycline. *Streptococcus viridans* were resistant to 24 (57%) of Kanamycin and 19(45%) of Gentamycin. *Staphylococcus Epidermidis* and *Staphylococcus Aureus* were present in 6 (11 %) and 3 (6 %) cultures, respectively. 3 (6 %) of the pure cultures produced anaerobic growth. 2 of the 5 cultures specifically submitted for anaerobic culture produced anaerobic growth, and in both cases the species was *Actinomyces.* Mixed cultures (revealing more than 1 organism) accounted for 8 (12 %). A lesser degree of resistance was noted among the *Staphylococci* to tetracycline, erythromycin, ampicillin, kanamycin, gcntamicin, methacillin, and clinclamycin.

Bartlett and Gorbach in 1975[49] conducted a study among a total of 84 patients, with aspiration pneumonia and primary lung abscess. 49 patients were treated with pencillin G and 35 were treated with clindamycin. The predominant organisms were *peptostreptococci, peptococci, B.Melaninogenicus, fusobbacteria and bacteroids fragilis,* majority of the anaerobic bacteria were sensitive to pencillin G. There were only 2 failures among 49 patients receiving pencillin G. Clindamycin were equally effective in anaerobic pulmonary infections. Author pointed out, it is not necessary to eliminate every

bacterial species in mixed cultures to achieve the result. And showed the example of infections where the penicillin therapy remains effective even in the presence of penicillin resistant *Bacteriodes fragilis*. This is due to the fact that by eliminating the other pathogens; the microenvironment is altered making it favorable for penicillin. Authors further added that successful treatment of infection depend as much on the environment either by the debridement or drainage of the infected tissue as well as by antimicrobial usage aimed at eradicating the causative organisms.

A. G. HELSTAD et al in 1977 conducted a comparative study evaluating the ability of the three widely used transport systems: (i) aspirated fluid in a gassed-out tube (FGT), (ii) swab in modified Cary and Blair transport medium (SCB), and (iii) swab in a gassed-out tube (SGT), For maintaining the viability of aerobic anaerobic and facultative bacteria. 25 aspirated specimens from clinical infections cultured and evaluated the recovery of anaerobic, aerobic, and facultative bacteria in transport tubes were held at 25°C and semi-quantitatively sampled at 0, 2, 24, and 48 hours. yielded 75 anaerobic strains and 43 isolates of facultative and 3 of aerobic bacteria. Only one anaerobic isolate was not recovered in the first 24 hours only in the SGT. At 48 hours, 73 anaerobic strains (97%) were recovered in the FGT, 69 (92%) in the SCB, and 64 (85%) in the SGT.

Robin Woods in 1978[34] reviewed the current antibiotic sensitivity patterns of bacteria isolated from 140 cases of acute dental pyogenic infections and the types of bacteria associated with these infections and their antibiotic sensitivity patterns are discussed and related to earlier reports. The incidence of antibiotic allergy, the principal contraindication to the use of certain antibiotics, has been assessed and appropriate alternative antibiotics are considered. And further, to evaluate several new

chemotherapeutic preparations. He found that the most frequently isolated bacteria from pyogenic lesions are streptococci (80 %). There were also a substantial number of (13.6 %) staphylococci, and 6.4 % of Gram negative bacteria. Penicillin remains the first choice for treatment of streptococcal infections and for those for whom penicillin is contraindicated, erythromycin is the alternative. The penicillinase resistant penicillin cloxacillin is the treatment of choice for staphylococcal infections, although co-trimoxazole appears more effective. Cephalosporins are effective against staphylococci and Gram negative bacteria as well as most of streptococci encountered in the dentistry.

Boon JR et al in 1982 conducted an experimental study and found out the benefits of adding clavulenic acid to amoxicillin. The distribution of amoxicillin clavulenic acid combination in infected animals after administration of amoxicillin clavulenic acid was evaluated by the measurement of concentrations of substances present in the specimens collected at the sites of infection and showed that both the amoxicillin and clavulenic acid is well distributed in the animal body and present in significant concentrations at various sites of infection. The ability of clavulanic acid to protect amoxicillin in vivo was confirmed by the efficacy of amoxicillin-clavulanic acid formulations in the treatment of infections.

James J. Crawford[35] et al in 1983 described about the Infections of the Orofacial Tissues, types of Infection, and the types of Bacteria in Pyogenic Orofacial Infections, infection Management, and choosing the Antibiotic Therapy. Most bacteria in odontogenic or oral infections belong to anaerobic species includes a oral streptococci and anaerobic species, especially the *Bacteroides, Fusobacterium,* anaerobic cocci, and *Actinoniyces* species, are the most common agents of pyogenic submucosal orofacial

infections. Management includes drainage and debridement. The Decisions to use Antibiotics, duration of therapy, the reasons of Antibiotic therapy failure and the Common Errors in Antibiotic Therapy were described.

Michael A. O. Lewis et al in 1988 reported that the antibiotic susceptibility of 50 acute dento-alveolar abscesses were determined by testing of the primary cultures of pus and secondary cultures of individual isolates, they used a comparative disc method. The sensitivity reports obtained by the primary testing agreed with that of secondary tests for 47 (94%) of the abscesses were studied. primary testing of pus samples aspirated from acute dentoalveolar abscess is reliable and can provide the clinician with antibiotic sensitivity results more rapidly than conventional secondary testing, especially when slow growing anaerobes are involved. Which yielded a total of 166 bacterial strains was studied. 4 of the specimens were polymicrobial with a mean of 3.3 bacterial strains per sample; 20 (40%) of the abscesses revealed strict anaerobes, 3 (6%) revealed facultative anaerobes, but 27 (54%) revealed a mixture of both types of bacteria. Since slow-growing strict anaerobes, particularly *Bacteroides* species and anaerobic gram-positive cocci, were frequently isolated from acute dentoalveolar abscess a reliance on purely secondary sensitivity testing would result in a delay of 4-5 days before susceptibility information could be available.

Y. Gill and C. Scully in 1990[14] described that, anaerobes play a major role in odontogenic infections and the most common microbial isolates are Bacferoides, fusobacteria, peptococci, peptostreptococci and viridans streptococci. Drainage should be established where possible. Penicillin is still the drug of first choice with metronidazole a good alternative. Nevertheless, not all clinicians are aware of current views and,

therefore, this article is a state-of-the-art review for practicing clinician of microbiology and antimicrobial therapy of orofacial odontogenic infections.

M.A.O. Lewis et al 1990[12] examined the findings of microbiological studies of acute dento-alveolar abscess and identified the likely pathogens and their sensitivity to antimicrobials. He found that the microbial flora is usually polymicrobial in acute dento alveolar abscess involving strictly anaerobic Gram- positive cocci, facultative anaerobic Gram- positive cocci, and strictly anaerobic Gram-Negative bacilli. Choice of antimicrobial therapy is phenoxy methylpencillin 250mg 6^{th} hourly, for those with hypersensitivity to pencillins, erythromycin 250 mg 6^{th} hourly or amoxicillin 250mg 6^{th} or 8^{th} hourly, recommendation of alternative therapy such as metronidazole, orinidazole, cefotetan, cefadroxil, clindamycin, and high dose amoxicillin if isolation of occasional strains of pencillin resistant Bacteroides species has resulted.

Brook I et al in 1991[8] conducted a study which includes 39 patients with periapical abscess. Aspirates of pus were taken and studied for aerobic and anaerobic bacterial growth. Bacterial growth was presented in 32 specimens includes 78 isolates (55 anaerobic and 23 aerobic and facultative organisms). Predominant organisms identified are Streptococcus spp (20), Bacteroides spp. (23 isolates, including 13 Bacteroides melatiinogenicus group)., anaerobic cocci (18), and fusobacterium spp (9). Beta-lactatnase-producing organistns were recovered and suggest that the polymicrobial nature and presence of anaerobic bacteria in periapical abscess. 33% of beta-lactamase in the abscess reveals that resistance of pencillin and the requirement of other antimicrobials such as clindamycin chloramphenicol, carbencillin, cefoxitin imipenem.

Harold C. Neu et al in 1992 [20] reported about the mechanism of resistance and mechanism of action of different anti microbial agents and their action on aerobic and anaerobic species. He describes that the Bacteria have become resistant to antimicrobial agents as a result of the exchange of genetic material via plasmids and transposons or chromosomal changes. Mechanisms of resistance of Streptococcus pneumoniae, Streptococcus pyogenes, staphylococci, Enterobacteriaceae and Pseudomonas families, Haemophilus influenza, Neisseria and Moraxella, Enteric Pathogens and Anaerobic Bacteria to antimicrobials were described. Mechanisms to overcome the bacterial resistance ranged from obvious hygienic practices to stop the spread of bacteria and to the synthesis of agents with improved antimicrobial activity should be adopted in order to limit the bacterial resistance.

Vejayan Krishnan et al in 1993[4] described the management of potentially life-threatening infections and to establish the protocol for the treatment. This study reviewed 50 infections treated over a 3-year period. The most frequent cause for the infection was odontogenic (43 patients). Clindamycin was the antibiotic of choice used at dosage of 900 mg every 8 hours IV or 300 mg every 6 hours orally and it was used in 36 patients. Penicillin.G was used in dosages of 2 to 4 million units every 4 to 6 hours intravenously or 500 mg every 6 hours orally in the remainder of patients. Alpha-hemolytic *Streptococcus* was the most commonly isolated organism. Resistant organisms have developed due to its long and widespread use. Clindamycin became preferred antibiotic for empiric therapy in this study. A protocol for the management of maxillofacial infections is described. The results revealed rapid resolution of the infections by adhering

to fundamental principles of management: recognition of airway compromise, surgical intervention, and the administration of the appropriate antibiotic.

Daniel H. Fine et al in 1994 described the importance of the microbial surveillance is explained in 3 clinical cases. Each of the case demonstrated a continued lack of response to conventional periodontal therapy. Repeated bouts of periodontal abscess formation and bone loss have occurred over a 3 to 4 year period, despite numerous surgeries supplemented with antibiotics. As a result, patients were termed refractory to treatment and extensive microbiological analysis and sensitivity testing was done, Actinobacillus actinomycctemcomitans was identified and Tetracycline resistant, Amoxicilhn sensitive, Actinomycetemcomitans was discovered from first case, Temafloxicin led to elimination of Staphylococcus aureus in second case. And in the third case reveals the poiymicrobial nature of the disease-associated plaque would most probably have been uncovered eventually by the newer methods such as fluorescent antibody identification and DNA probe identification (FA and DNA probes). Following Administration of the appropriate antibiotic and conservative therapy consist of several sessions of scaling and root planning, each of the cases demonstrated a dramatic remission of disease progression. No further breakdown has been seen for a minimum period of 2.5years. These cases support the usefulness of microbial identification coupled with antibiotic sensitivity as an adjunct to conventional conservative periodontal therapy.

Gady Har-El et al in 1994[9]conducted retrospective study on 110 patients with diagnosis of different types of abscess. The most common type of organisms involved are, Streptococcus viridians (40.9%), Staphylococcus aureus (27.3%), Streptococcus epidermidis (22.7%) and Bacteriodes (17%) and anaerobes accounts for 40% of the

positive cultures were Bacteroides. Group-A Beta-Hemolytic streptococci were found in 6.8% and gram-negative aerobic organisms such as Escherichia coli Proteus, and Pseudomonas were found in less than 6% of cultures, the most common regimen was penicillin G and an anti-staphylococcal drug. Clindamycin or metronidazole was used in penicillin allergic patients. Other combinations or a single broad spectrum drug therapy, such as, ticarcillin was used in less than a third of the patients.

Bridgemen A. et al in l995[13] reviewed 107 cases of acute maxillofacial infections, aerobic and anaerobic microbiological study has been conducted in 81 cases, of which 98% yielded growth, majority were mixed infections. Mixed oral flora including aerobic and anaerobic streptococci alpha-haemolytic streptococci, Bacteroides, Actinomyces israelii Methicillin-resistant Staphylococcus aureus (MRSA) was the only isolate in five cases, beta-lactam and a nitroimidazole were the most commonly adopted regimen, crystalline penicillin 2 million units 4 to 6 hourly with metronidazole500 mg 8 hourly. Antimicrobials were prescribed empirically at the time of presentation and altered according culture and susceptibility test results, and dependent on the patient's progress. Authors stated that penicillin is the drug of choice, because it is inexpensive, and bactericidal for majority of bacterial strains in odontogenic infections. Also stated that bacterial resistance to penicillin has emerged among the strains of alpha haemolytic Streptococci, Bacteriodes species and therefore has encouraged the use of metronidazole. They also quoted that in current series 81 (75.7%) patients received combination of penicillin and metronidazole.

D. M. Livermore et al in 1996[44] from ICU patients at 35 centers were collected 966 isolates of Consecutive klebsiellae. Isolates were sub-cultured on MacConkey agar to

confirm purity, and its identity was checked. Those confirmed as klebsiellae were stored at 20°C on nutrient agar slants. Out of 966 isolates, 716 were Klebsiella pneumoniae, 248 were Klebsiella oxytoca and 2 were Klebsiella ozaenae. Production of extended-spectrum beta-lactamases (ESBLs) was derived in 220 isolates on the basis of synergy between ceftazidime and clavulanate. Putative ESBL producers received from 23 centers, including 20 of 27 that contributed more than 10 klebsiellae. Over 88% of putative ESBL producers were resistant to ceftriaxone 1 mg/L ceftazidime 2 mg/L, and aztreonam 1 mg/L, whereas, amongst ESBL-negative isolates, more than 98% of the K. pneumoniae and 87% of the K. oxytoca were susceptible to the above concentrations. Putative ESBL producers were more resistant to cefuroxime and cefoxitin than non-producers, but not to biapenem. MIC distributions of piperacillin/tazobactam, aminoglycosides and ciprofloxacin were bimodal for ESBL producers, with some isolates highly sensitive and others were very resistant. 70% of putative ESBL producers were susceptible to piperacillin/tazobactam 16+ 4 mg/L, but 30% were resistant, some were highly so. Resistance to this combination, and to the ciprofloxacin, was clustered in certain centers. 2 other groups of cephalosporin-resistant isolates were also identified besides ESBL producers, viz. (i) 20 K. oxytoca, from 15 centres and (ii) nine isolates, from three centers. Examination of the hospitals' own susceptibility data indicated up to 33% of putative ESBL producers had been reported susceptible to the third-generation cephalosporins or monobactams.

Papapanou P.N. et al in 1997 compared the routine culture techniques of non-selective and selective media in combination with routine biochemical testing using commercial test panels with "checkerboard" DNA-DNA hybridization methodology

using whole genomic digoxigenin labeled DNA probes for the analysis of the composition of subgingival microbiota. 70 subjects with a total of 283 subgingival plaque samples analyzed for Porphyrotnonas gingivalis. prevotella nigrescens, prevotella intermedia, bacteroides forsythus, fusobacterium nucleatum, campylobacter rectus, eikenella corrodens, actinobacillus actinotnycetemcomitans, streptococcus mutans and streptococcus sanguis. Checkerboard technology has resulted in higher prevalence figures for half of the species tested when compared to culture data. Also the checkerboard methodology has resulted in statistically significant higher bacterial counts for the majority of the species.

M. Sakaguchi et al in 1997[21] did retrospective study on 91 patients with deep neck infections to determine the pattern of clinical disease and to formulate a management plan. The aim of the study was to identify the pattern of clinical condition and to formulate a management plan. Spaces involved were determined by clinical, radiologic, and by operative findings were peritonsillar (72), parapharengeal (8), submandibular (7), retropharengeal (1), superficial (1), anterior visceral space (1) and visceral vascular space (1). Gram's stain, anaerobic and aerobic cultures, were done on purulent material obtained by the aspiration with a syringe or by swabs. Results of cultures were only available for 29 of the 91 patients. 9 Of these, 25 cultures were positive and 4 were negative. 12 of the 25 specimens had more than 1 organism. Most common organisms isolated were streptococci viridans group, followed by Neisseria species and anaerobes accounted for 4/25 (16%) of the positive cultures. He concluded that the treatment of deep neck infections has three main aspects: medical management, surgical management, and airway control.

Franklin D. Lowy et al in 1998 described extensively about the staphylococcus aureus organisms and medical progress of staphylococcus aureus infections includes its components and products. Pathogenesis of the disease, mechanisms of resistance to antimicrobial agents, treatment of staphylococcus aureus infection, prevention of the disease were described. Also described about the frequencies of both hospital and community acquired staphylococcus infections, the frequency of infection increases steadily and treatment became more difficult because of the emergence of multi drug resistant strains.

Keith F. Barker in 1999[18]conducted a review over antibiotics used in United Kingdom and the antibiotic resistance in British population. Author states that mechanism of resistance in bacteria is mainly due to (a) Enzymatic modification or destruction of the antibiotic; (b) Reduced antibiotic uptake into the bacterium; (c) Increased efflux of antibiotic from the bacterium; (d) Alteration or production of a new target site; (e) Over-expression of the drug target site. Author concluded that the bacterial infection remains a public health problem worldwide over past 50 years with the development of antibiotic resistance. In recent years multi-resistant gram negative bacteria 'superbugs' has been reported and justified in their studies. Author also concluded that antibiotic resistance is a complex, continually evolving problem which is often difficult to put into perspective. Highly simplistic approaches to reduce the consumption and develop significant new agents are formidable tasks.

Tomoari Kuriyama et al in 2000[50] conducted a study and the aim of the study was to determine the current status of beta-lactamase producing bacteria in orofacial odontogenic infections. Microbiologic data regarding the purulent exudates from 111

cases with orofacial odontogenic infections were analyzed in relation to past administration of beta -lactams. Beta-lactamase producing bacteria were isolated more frequently from the beta -lactam administered group (38.5%) than from the beta -lactam-nonadministered group (10.9%; $P < .005$). The predominant bacteria isolated included *Prevotella* (the most frequent isolate), streptococci viridans, *Fusobacterium*, *Peptostreptococcus*, and 7.1% of total isolates produced beta-lactamase. Penicillin and cefazolin worked well with beta-lactamase–nonproducing *Prevotella.* Cefmetazole, sulbactam/cefoperazone, and imipenem worked well against both types of *Prevotella*. If the patient has not received beta-lactam antibiotics in course of infection, even if the patient has received β-lactam antibiotics with an appropriate dose for a deviation of l day or 2 days, the prescription of penicillin and first generation cephalosporins is suitable. If the patient already received antimicrobial therapy with β-lactams in the course of infection for duration of 3 days or more, in such cases beta lactamase-stable beta- lactam are recommended.

Tomoari Kuriyama et al in 2000 conducted a study on 163 patients with orofacial odontogenic infection and concluded that *Streptococci viridians*, anaerobic gram–positive cocci and anaerobic gram-negative rods are isolated frequently from the orofacial infection. The susceptibility results suggest cefazolin may not have more advantages than penicillin, but cefmetazole may be more effective against infection than the penicillin. Cefmetazole were effective against all test pathogens. Erythromycin was ineffective against streptococcus viridans and most *Fusobacterium*. Clindamycin exerted a strong antimicrobial activity against anaerobes. Penicillin was effective against almost all the pathogens, although it did not work well against β-lactamase positive Prevotella.

Minocycline were effective against most of the test pathogens. The antimicrobial activity of levofloxacin against viridans streptococci was not strong.

Kuriyama T.et al in 2001 conducted a study from 93 pus specimens of orofacial odontogenic infections. The incidence of beta lactamase production in anaerobic gram negative rods isolated and antibiotic susceptability of these isolates against 11 antibiotics were determined. 191 anaerobic gram negative rods were isolated, beta-lactamases was detected in 35.6% of the black pigmented prevotella and 31.9% of the non pigmented prevotella. Ampicillin, cefotaxim, and cefazolin showed decreased activity. Where as the activity of ampicillin/sulbactum, imipenem and cefmetazole continued to be effective against beta lactamase positive prevotella strains. All tested beta lactamase antibiotics where effective against *Porphyramonas* and *Fusobacterium*. Erythromycin showed decreased activity against nonpigmented *Prevotella* and *Fusobacterium*. Minocyclin, clindamycin and metronidazole were powerfull antibiotics against which anaerobic gram negative rods could be tested.

William Storoe in 2001[11] performed a retrospective review, which compared two cohorts of the patient admitted in 1980 and in 1990. He found there is a marked difference in type and prevalence of bacteria isolated. Gram positive cocci were frequently found in 1990s patient than from the 1980s patients. 1990s patients were more resistant to antibiotics compared to 1980s patients. There were no clinically significant differences between patient characteristics admitted during 1980 and 1990. Although there were differences in type and prevalence of bacteria isolated, it was mainly due to changes in nomenclature and identification protocols and isolation techniques.

Kuriyama T et al in 2002[2]. Described the beta-lactamase production and antimicrobial susceptibility of mainly anaerobic gram-negative rods were isolated from the pus specimens of 93 orofacial odontogenic infections and reported the bacteriology and antimicrobial susceptibility of the bacteria other than anaerobic gram-negative rods, mainly gram-positive cocci, *Peptostreptococcus micros and Streptococcus constellatus* were the frequent isolates. *Eubacterium* species, *Corynebacterium* species, and *Peptostreptococcus prevotii*, were recovered only from dentoalveolar infections, *Gemella morbillorum* were found more frequently in periodontitis than in other infections. Beta-Lactamase-positive strains were detected only in staphylococci. Ampicillin, ampicillin/sulbactam, cefazolin, cefotaxime, imipenem, erythromycin, clindamycin and levofloxacin showed high susceptibility rates (77%) against viridans streptococci, *Peptostreptococcus* and *Gemella*. Minocycline showed a high MIC_{90} value against viridans streptococci (32 mg/ml), and metronidazole was effective against *Peptostreptococcus* and *Gemella*.

Flynn TR et al in 2003[17], described about the antibiotic selection in the head and neck infections. He described about the molecular biology of the antibiotic resistance. The bacteria acquire antibiotic resistance by alteration of a drugs target site, inability of a drug to reach the target, inactivation of the antimicrobial agent, active elimination of the antibiotic from the cell. The bacteria acquire resistance genes by spontaneous mutation, gene transfer, bacteriophages, mosaic genes, and described about the various antibiotics.

Saini. S. et al in 2003[16] conducted a study and compared the normal aerobic and anaerobic oral flora with flora from the deep seated dental caries, gingivitis, and periodontitis. All 100 cases include 25 control groups yielded microbes. 97% of orodental

infections are polymicrobial, and three or more microbes were found in 84% of the cases. Streptococcus mutans and anaerobic lactobacilli were common in the dental caries; Actinomyces and Peptostreptococcus species, were found in gingivitis. Actinobacillus actinomycetemcomitans and porphyromonas gingivalis were found in periodontitis.

D.K Dhariwal et al in 2003 reported a rare and potentially fatal complication of epidural spinal abscess following a dental extraction, the causative organism was Streptococcus *milleri* for which Cefotaxime and flucloxacillin were given intravenously.

Richard H. Haug in 2003 reported about the changing microbiology of maxillofacial infections. The changes in the microbiology of infections of odontogenic etiology rest in changes in nomenclature and ability to isolate the organisms. The changes in the microbiology of infections associated with maxillofacial trauma are not caused by the injuries but are related to the nosocomial systemic infection and emergence of the resistant microorganisms. The changes in the microbiology of nasal and paranasal sinus infections are associated with the emergence or reemergence of strains of bacteria that are resistant to the common antibiotics.

Stefanopoulos P. K. et al in 2004[45] reviewed the literature on orofacial odontogenic infections, and indicates that the underlying microflora is polymicrobial, predominantly involving strictly anaerobic gram-positive cocci and gram-negative rods, along with facultative and microaerophilic streptococci. Superoxide dismutase produced by moderate anaerobes renders them by definition tolerant of oxygen levels of 2% to 8% and is considered a prerequisite to their pathogenicity. Antibiotic resistance is an important consideration in managing the orofacial odontogenic infections. Reported that

the Beta-lactamase activity among gram-negative anaerobic rods aer responsible for clinical failures with penicillin treatment. Although penicillin remains the antibiotic of choice for mild to moderate odontogenic infections in the immunocompetent host but pencillin should not be used as initial therapy for more serious infections possibly involving penicillin resistant oral anaerobes. However, in more severe cases where there is a narrow margin of acceptance of possible therapy failure, it is recommended to manage with adequate anaerobic spectrum such as clindamycin or combinations of an aminopenicillin with beta-lactamase inhibitor; broader antimicrobial coverage is indicated for patients with impaired host defenses.

J. Wang, et al in 2005 conducted a five-year retrospective study of odontogenic maxillofacial infections in a large urban public hospital. Out of 250 cases 157 cases of infection was odontogenic in origin. Only eight of the 157 patients were prescribed intravenous penicillin alone. 94 received penicillin plus metronidazole, while 31 were given clindamycin therapy, 14 a cephalosporin, and 10 a combination of antibiotics, usually gentamicin, ampicillin and metronidazole, this being reserved for the most severe infections. Authors identified potential risk factors and suggested that early dental extraction, incision and drainage, coupled with intravenous antibiotic therapy, is the most effective treatment. Antibiotic therapy can be empirical. Obtaining of cultures and sensitivity reports does not appear to be clinically helpful, and did not lead directly to any antibiotic or other treatment changes.

Boyanova L. et al in 2006 conducted a study and evaluated the incidence and susceptibility to antibacterial agents of anaerobic strains in 118 patients with head and neck abscesses (31) and cellulitis (87). Odontogenic infections were the most common

source in 73 of (77.7%) of 94 patients. Anaerobes in patients with odontogenic and other sources of infection were 82.2 and 71.4%. Total of 174 anaerobic strains were found. The predominant bacteria were *Prevotella* (49 strains), *Fuso bacterium* species (22), *Actinomyces* spp. (21), *Anaerobic cocci* (20) and *Eubacteriurn* spp. (18). *Bacteroides fragilis* strains isolated from 7 (5.9%), specimen. The resistance rate to amoxicillin of Gram-negative anaerobes was 26.9% (21 of 78 strains). Resistance rates to clindamycin and metronidazole of Gram-negative anaerobes were 5.4% (4 of 74) and 2.5% (2 of 79), respectively, and those of Gram-positive species were 4.5% (3 of 66) and 58.3% (42 of 72), respectively. Only one strain was not susceptible to ampicillin/sulbactam.

Thomas R. Flynn et al in 2006[22] evaluated 37 consecutive hospitalized patients with odontogenic infection were treated with intravenous penicillin and prompt incision and drainage. Seventeen (19%) of the 90 isolated strains were penicillin resistant; one or more penicillin resistant organisms were found in 13 (54%) of the 24 cases with antibiotic sensitivity data. 4 clindamycin-resistant strains were identified, one each of *Streptococcus milleri*, *Eikenella corrodens*, and *Streptococcus mitis*, and one strain of *Klebsiella pneumonia* that was also resistant to penicillin. Clindamycin-resistant strains were identified in 4 (17%) cases with sensitivity data. Author concluded that penicillin resistance, resulting in penicillin therapeutic failure, was unacceptably high in this sample. Alternative antibiotics, such as clindamycin, should be considered in hospitalized patients with odontogenic infection.

Thomas R. Flynn et al In 2006 conducted a study on thirty seven consecutive patients and identified the significant predictors of four outcomes in patients with severe odontogenic infections: penicillin therapeutic failure (PTF), abscess formation, need for

reoperation and length of hospital stay (LOS). Found that culture of *Peptostreptococci* was a negative predictor of abscess formation. There was no significant predictor of PTF or reoperation on multivariate analysis, although Pencillin-resistant organisms were isolated in all cases of PTF. Author concluded that the increased LOS in severe odontogenic infections is predicted by the anatomic extent and severity of the infection and the occurrence of complications such as PTF and the need for reoperation. PTF is significantly associated with later identification of PCN-resistant organisms.

Rege A.J. et al in 2006[10] conducted a six year retrospective study on 103 patients. The submandibular space was most frequent for single space abscesses (30%) followed by buccal space (27.5%) and lateral pharyngeal space (12.5%). The most common bacteria isolated were streptococci, *Prevotella*, *Staphylococci* & *Peptostreptococcus*. *Streptococci viridans demonstrated* an 87.1% sensitivity rate to penicillin. *Streptococci Viridans* also exhibited high susceptibility to ampicillin (98.4%), clindamycin (86.3%), ciprofloxacin (100%), levofloxacin (98.6%) cefazolin (100%), erythromycin (83.4%), and vancomycin (100%). *Staphylococci* showed a 27.3% susceptibility rate to penicillin and sensitivity to ampicillin (41.2%), cefazolin (70.0%), ciprofloxacin (95%), clindamycin (89.5%), erythromycin (75%), levofloxacin (84.2%), and vancomycin (100%).

Osborn T. M. et al in 2008 described about the anatomy of the fascial layers, deep neck spaces, microbiology of the deep neck spaces which includes the most common organism associated with deep neck infections was Staphylococcus aureus. Drug resistance has, however, contributed to a change in the microbial flora associated with these serious infections, which are now most commonly associated with *aerobic*

*streptococcal speci*es and nonstreptococcal *anaerobes.* Commonly cultured organisms include *Streptococcus viridans, Streptococcus milleri group species, B-hemolytic streptococci, Neisseria species, Peptostreptococcus, coagulase-negative staphylococci and Bacteroides.* Anaerobic bacteria include *Prevotella and Porphyromonas species, Actinomyces species, Bacteroides species, Propionobacterium, Hemophilus, and Eikenella.* Anaerobic bacteria are found in more than half of severe odontogenic infections.

Munish Kohli et al in 2009[47] identified the most common micro-organisms causing odontogenic infections and their antimicrobial susceptibility. from 80 patients pus samples were collected aseptically by aspirating the abscess using sterile 18/22 gauge needle with a 2ml syringe, the specimen was immediately inoculated in sterile Robertson cooked meat broth (RCM) for transportation of anaerobic organisms. They were cultured (aerobically and anaerobically) and stained for morphological study of the isolates. A total of 109 micro-organisms were isolated. In 28(35%) of cases pure aerobes were identified, pure anaerobes in 18(22.5%) cases, mixed aerobes and anaerobes in 10(12.5%) of cases, mixed aerobes in 15(18.75%) and mixed anaerobes were identified in 6(7.5%) cases. Ofloxacin was the most sensitive drug followed by ciprofloxacin and sparfloxacin for pure gram positive organisms. The most resistant drugs were amoxicillin and ampicillin. The gram negative colonies were sensitive to cefotaxime.

D. Robertson et al in 2009[1] published the microbiology of acute dental abscess and found that it is polymicrobial in nature and comprising facultative anaerobes, such as viridans group streptococci and the Streptococcus anginosus group, with predominately anaerobic cocci such as *Prevotella* and *Fusobacterium* species. Other causative

organisms found were *Bulleidia extructa*, *Cryptobacterium curtum* and *Mogibacterium timidum*.

Paolo Boscolo-Rizzo et al in 2009 conducted a retrospective study of clinical characteristics and the management of submandibular space infections and to identify the predisposing factors of life-threatening complications. Author concluded that airway obstruction and spread of the infection to mediastinum are the most troublesome complications of submandibular space infections. Patients with cellulitis and small abscesses can respond to antibiotics alone. Surgical drainage should be performed in patients with large abscesses, Ludwig's angina, anterior visceral space involvement, and in those who do not respond to antibiotic treatment. Early surgical drainage should always be considered even in seemingly less critical cases.

Fábio Ricardo Loureiro Sato et al in 2009 conducted a retrospective study in 210 patients. The main origin of infection was odontogenic (79.31%); The main facial spaces affected were the buccal mandibular space (50.00%), submandibular space (31.90%), and buccal maxillary space (19.05%). Surgical drainage were carried out in 46.67% of cases, and 10.95% of these interventions were performed under general anesthetic. Author concluded that no predisposition concerning gender or race was detected. The therapeutic protocol adopted presented very positive results, with a small number of complications.

Opeyemi O et al in 2009[23] reviewed their experiences with deep neck abscesses and identified the unique trends in their patient population with 106 Case series and chart review of patients with deep neck space abscesses, odontogenic infections were the most

common cause of deep neck abscesses (49.1%). Among the 52 odontogenic cases, *Streptococcus spp* were the most common pathogens (44.4%). Second or third generation cephalosporins such as cefoxitin or ceftriaxone were effective. Alternatively, a penicillin and beta-lactamase inhibitor combinations such as ampicillin-sulbactam also provides adequate coverage. Clindamycin used as an alternative and is preferred in those allergic to penicillin. Suspicion or confirmation of methicillin-resistant *Staphyloccocus aureus* warrants the use of effective antibiotics such as vancomycin, rifampin, or sulfamethoxazole-trimethoprim.

Mrudula Patel et al in 2009 conducted a study and compared the clinical, microbiological, enzymatic, and host immune response variables between 15 patients with Ludwig's angina and 42 patients without Ludwig's angina. Most Common organisms found are *Staphylococcus aureus* and black-pigmented bacteroides from patients with Ludwig's angina. Author concluded that the elevated levels of CRP and urea could indicate the severity of infection in patients with Ludwig angina.

Laith Hussein Al-Qamachi et al in 2010[30] stated that primary treatment for deep neck spaces odontogenic infection (DNSOI) with suppuration is surgery and systemic antimicrobial therapy. Initial antimicrobial therapy is empirical. Over the last decade, there is a change in practice with 2nd generation cephalosporins and metronidazole, replacing benzylpencillin and metronidazole. Recently, evidence has emerged suggesting that the antimicrobial resistance in nosocomial infections could be related to the widespread use of second and third-generation cephalosporins. A total of 75 cases were retrospectively identified, *Streptococcus milleri* and mixed anaerobes were predominant. Only in 3 cases (4%) were found to be penicillin-resistant. The substitution of

benzylpenicillin for cefuroxime as an initial empiric therapy for DNSOI has been equally efficacious in the large majority of cases.

Dipesh D. Rao et al 2010 performed a 4-year prospective study on maxillofacial space infections of odontogenic origin in diabetic patients compared with nondiabetic patients. Result showed that a total of 111 patients were identified out of which 31 were diabetic. The submandibular space being the most common space involved in both the group and organisms commonly isolated were *Streptococcus* species s. The empirical antibiotic used were amoxicillin plus clavulanic acid combined with metrogyl in 70.27% cases. Author concluded that *Streptococcus* species is still the most common causative pathogen irrespective of the diabetic status of the patient. The same empirical antibiotic therapy of amoxicillin plus clavulanic acid combined with metrogyl along with hyperglycemia control and surgical drainage of infection yielded satisfactory resolution of infection in the diabetic patients as well.

Paul. W. Poeschl et al in 2010[48] conducted a retrospective study and identified the major pathogens responsible for deep space head and neck infections and their current resistance to routinely used antibiotics. A total of 206 patients suffering from odontogenic deep space infections treated by means of surgical intervention and intravenous administration of antibiotics. The results showed that the predominant bacteria were *Streptococci viridans*, *Staphylococci*, *Peptostreptococcus*, *Prevotella*, and *Bacteroides*. The aerobes were resistant against clindamycin in 18%, macrolides in 14%, and penicillin G in 7%. The anaerobes were resistant to clindamycin in 11%, to metronidazole in 6%, and to penicillin G in 8%. Author concluded that the high

resistance rate for clindamycin and macrolides was especially striking and may necessitate an adaptation of our antibiotic regime in the future.

Marilyn E. Levi et al in 2011[15] Reviewed the serious nature and potential dangers that exists from odontogenic infections and the antibiotics available to treat these infections. Successful treatment requires an understanding of the microflora, the regional anatomy, and the disease process. In the oral cavity, more than 80% of the cultured bacteria include streptococci, Peptostreptococcus, Veillonella, Lactobacillus, Actinomyces and Corynebacterium.

Thomas R. Flynn in 2011[39] this article is an attempt to answer the questions with a systematic review of the currently available scientific literature on this multifaceted topic. The results of this systematic review may allow oral and maxillofacial surgeons (OMS) to have less concern over the choice of antibiotic prescription in the management of odontogenic infections. Among the antibiotics commonly used for odontogenic infections (OI), it seems that no one antibiotic is clearly superior to all others. Antibiotics may therefore be chosen according to cost and safety, with individualized consideration of the patient's medical history. Surgical treatment, consisting of incision and drainage and removal of the odontogenic cause by extraction, endodontic therapy, or other means, is of primary importance.

Materials & Methods

The study was conducted in total of 142 patients having orofacial space infections attending the Out Patient Department of Oral & Maxillofacial surgery in Sree Mookambika Institute of Dental Science, Kulasekharam from September 2010 to September 2012. Pus samples and aspirates were collected aseptically from the patients for aerobic and anaerobic microbiological study. Proper medical/dental history, clinical signs and symptoms were recorded and relevant investigations were done.

SAMPLE SIZE

Minimum of 142 patients with head and neck fascial space infections of odontogenic origin are taken up for the study. This includes males and females with an age group of 5 years to 73 years.

SELECTION CRITERIA

Inclusion Criteria

- Patients with head and neck fascial space infections of odontogenic origin.
- Patient who had not taken antibiotics for the head and neck fascial space infections of odontogenic origin.

Exclusion Criteria

- Patient taken antibiotics at randomly for head and neck fascial space infections of odontogenic origin.
- Patient not willing to participate in the study.
- Patients with head and neck fascial space infections other than odontogenic origin.

- Patients with culture and sensitivity results show no growth.

ARMAMENTARIUM

1. Local anesthetic lignocaine with adrenalin (LIGNOX 2% A, INDICO REMEDIES LTD. Mumbai, India)
2. Sterile 24 gauge and 25mm length needle with 3 ml syringe (Dispo Van, Hindustan Syringes & Medical Devices LTD. India)
3. Sterile 18 gauge and 38mm length needle with 5 ml syringe (Dispo Van, Hindustan Syringes & Medical Devices LTD. India)
4. Bard Parker blade number-11 (GLASSVAN sterile surgical blades, Niraj Industries Pvt. Ltd, Faridabad, India.)
5. Bard Parker Handle (Sirag surgical Enterprises, Chennai, India)
6. Transport cotton swab stick (Labtech medico Pvt. Ltd. Kerala, India)
7. Brain heart infusion broth (HIMEDIA Laboratories Pvt. Ltd. Mumbai India.)
8. Chocolate agar plate (HIMEDIA Laboratories Pvt. Ltd. Mumbai India.)
9. Maconkey's agar plate (HIMEDIA Laboratories Pvt. Ltd. Mumbai India.)
10. Nutrient Agar plate (HIMEDIA Laboratories Pvt. Ltd. Mumbai India.)
11. Blood agar plate (HIMEDIA Laboratories Pvt. Ltd. Mumbai India.)
12. Mueller Hinton Agar plate (HIMEDIA Laboratories Pvt. Ltd. Mumbai India.)

13. Antimicrobial disc. (Labtech medico Pvt. Ltd. Kerala, India)

14. Anaerobic jar (HIMEDIA Laboratories Pvt. Ltd. Mumbai India.)

15. Anaerogas pack with indicator tablet (HIMEDIA Laboratories Pvt. Ltd. Mumbai India.)

ANTIBIOTICS USED FOR THE STUDY

S. No	Antimicrobial Class	Representative Antibiotics
1.	Sulfonamides	Sulfadiazine
2.	Fluoroquinolones	Ofloxacin, Norfloxacin Gatifloxacin, Levofloxacin, Nalidixic acid, Ciprofloxacin, Cotrimoxazole
3.	Aminoglycoside	Gentamycin, Amikacin, Neomycin, Streptomycin
4.	Macrolides	Erythromycin, Azithromycin , Roxithromycin
6.	Penicillin's	Ampicillin, Amoxicillin, Penicillin G, Amoxicillin and clavulanic acid, Piperacillin, Cloxacillin, Meropenem
7.	Cephalosporin	Cefotaxime, Cefixime, Cefuroxime, Cefpodoxime Cephalexin, Ceftazidime, Cefazolin
8.	Broad Spectrum Antibiotics	Doxycycline, Tetracycline, Chloramphenicol
9.	Miscellaneous antibiotics	Linezolid, Clindamycin Bacitracin, Vancomycin, Furoxone, Nitrofurantoin, Septran, Sporidex

PROCEDURE FOR AEROBIC CULTURE

STEP 1 -SAMPLE COLLECTION

- After wearing sterilized gloves, site was anesthetized by local anesthetic (2% lignocaine with adrenalin) depending on the condition, the pus sample either

collected intra orally or extra orally. Pus sample were collected by transport cotton swab stick directly from the site and it was sent immediately for aerobic culture.

STEP 2 - SPECIMEN INOCULATION AND CULTURE

- Clinical material was transported immediately to the lab. The pus sample from the transport cotton swab stick is inoculated into the media plates by using a flame sterile straight wire loop to streak the inoculum into the media and spread the inoculum in a zig-zag pattern.
- The culture was done in the following medium.

1. Nutrient agar medium
2. Blood agar medium
3. MacConkey agar medium

NUTRIENT AGAR

Composition

Ingredients		**Gms / Litre**
Peptic digest of animal tissue	-	5.000
Sodium chloride	-	5.000
Beef extract	-	1.500
Yeast extract	-	1.500
Agar	-	15.000

Final pH (at 25°C) 7.4±0.2

Preparation- Add 28g of nutrient agar in 1000ml of distilled water. The mixture is then heat to boiling till the medium dissolve completely. Then the medium is sterilized by Autoclaving at 15 lbs pressure (121°C) for 20 minutes. Allow this solution to cool to ~ 50°C. It should be keep a close watch over the solution as it cools; if leave it too long, it will solidify in the jar and will be of no use. Do not pour the solution when it is too hot as it may damage the petri dishes. Pour solution into petri dishes and allow it to cool to room temperature to set. In most cases, the pouring of the agar should be done in the laminar flow hood to maintain sterile conditions. The Plates can be stored in the fridge for up to 4 weeks for use.

BLOOD AGAR

Composition

1. Peptose Peptone - 15.00 gm/litre
2. Liver extract - 2.50 gm/litter
3. Yeast extract - 5.00 gm/litre
4. Sodium chloride - 5.00 gm/litre
5. Bacteriological Agar - 15.00 gm/litre

   Final p^H (at 25^0 C) 7.4$\pm$ 2

Preparation: Suspended 21.25 grams of the medium in 500ml of distilled water and mixed well. It was heated with frequent agitation and boiled for 1 minute until the medium was completely dissolved. It was then distributed in appropriate containers. The medium was sterilized at 15 lbs pressure (121°C) for 15 minutes. Allow it to cool to

45°C-50^{0}C and aseptically added 7% sterile defibrinated sheep blood. Mixed well and poured into Petri dishes.

It is an enriched media, which is prepared by adding sheep blood (5-10%) to the sterilized melted nutrient agar at 45°C. It consist of nutrient agar 90 ml and sterile sheep blood 10 ml. it is one of the best laboratory media since most of the organisms grow well on it.

MACCONKEY AGAR

Composition

1.	Peptic digest of animal tissue	-	17.00 gm/litre
2.	Proteose peptone	-	3.00 gm/litre
3.	Lactose	-	10.00 gm/litre
4.	Bile salts	-	1.5 gm/litre
5.	Sodium chloride	-	5.00 gm/litre
6.	Neutral red	-	0.03 gm/litre
7.	Agar	-	15.00 gm/litre

Final pH (at 25^0 C) to 7.1$\pm$0.2

Preparation: Suspend 51.53 gm of powder in 1 liter distilled water. Mix well dissolves it by heating with frequent agitation. Boil for 1 minute until complete dissolution. Sterilize in autoclave at 121°C for 15 minutes cool to 45°C mix well and dispense into plates.

Allow the plates to solidify and place them upside down to avoid excess moisture on the surface of the medium. Prepared medium should be stored at 8-15°C. Colour is violet red. It is a culture medium designed to grow Gram-negative bacteria and stain them for lactose fermentation. The lactose fermenters form the pink colonies while non lactose fermenters produce colorless or pale colonies.

- Then these inoculated culture media plates were immediately incubated at 37^{o} C for 24 to 48 hours in an aerobic atmosphere, after 24 to 48 hours the plates are ready for the observation of colonies and further gram staining, biochemical and antibiogram tests.

GRAM STAINING

The gram staining reaction is used to identify the pathogens in specimens and cultures by their Gram reaction and morphology. Gram positive bacteria stain dark purple with crystal violet or methyl violet and are not decolorized by acetone or ethanol. Gram negative bacteria stain red because after being stained with crystal violet or methyl violet they are decolorized by acetone or ethanol and take up the red counter stain such as neutral red, safranin or dilute carbol fuchsin.

STEP 3- BIOCHEMICAL TESTS FOR IDENTIFICATION OF MICROBES

- After material is dispended to culture media and incubated for 24 to 48 hours will visualize colonies of grown organism. Then all sets of plates will apply following biochemical tests to identify the genus and species of bacteria they are as follows:-

    1. Methyl red test

2. Voges-Pros Kauer test
3. Oxidation fermentation test
4. Indole test
5. Triple Sugar Ion test
6. Uriase test
7. Citrate test
8. Manitol test
9. Motility test

After 24 hrs we will inspect the following test and diagnosis of infection is made.

METHYL RED TEST

This test is employed to detect the production of acid during the fermentation of glucose and maintenance of a pH below 4.5 in an old culture. Five drops of 0.04% solution of methyl red are added to the culture in glucose phosphate medium which had been incubated at 30 degree centigrade for five days, mixed well and a red colour is positive while yellow signifies negative test.

VOGES-PROS KAUER TEST

This test depends on the production of acetyl methylcarbinol from pyruvic acid, as an intermediate stage in its conversion to 2:3 butylene glycol. In the presence of alkali and atmospheric oxygen, the small amount of acetyl methylcarbinol present in the medium is oxidized to diacetyl which reacts with the peptone of the broth to give red colour. First alpha-napthol (also called Barritt's reagent A) and then potassium hydroxide (also called Barritt's reagent B) are added to the tube. The culture should be allowed to

sit for about 15 minutes for color development to occur. If acetoin was produced then the culture turns a red color (positive result); if acetoin was not produced then the culture appears yellowish to copper in color (a negative result).

OXIDATION FERMENTATION TEST

To perform this test simply swab some of your test culture into one of the boxes on an oxidase dry slide. If a color change to purple or blue is evident at 30 seconds to 1 minute, then the result is positive. It is important that the test is read by one minute to ensure accurate results. This laboratory test is based on detecting the production of the enzyme cytochrome oxidase by Gram-negative bacteria. It is a hallmark test for the Neiserria. It is also used to discriminate between aerobic Gram-negative organisms like Pseudomonas aeruginosa and other Enterobacteriacia.

INDOLE TEST

Testing for indole production is important in the identification of enterobacteria, most strains of E.coli, P.vulgaris, P.rettgeri, M.morganii, and providencia species break down the amino acid tryptophan with the release of indole. Red surface layer shows positive indole test and negative indole test shows no red surface layer.

URIASE TEST

This test is used to detect the enzyme urease, which breaks down urea into ammonia. Ammonia is a base and thus will raise the pH of the media if it is present. This change in pH is indicated by a pH indicator called phenol red which is present in the

media. A color change from yellow to bright pinkish-red is positive; lack of color change is a negative result.

CITRATE TEST

Citrate test is used test the ability of bacteria to convert citrate (an intermediate of the Kreb's cycle) into oxaloacetate (another intermediate of the Kreb's cycle). In this media, citrate is the only carbon source available to the bacteria. If it cannot use citrate then it will not grow. If it can use citrate, then the bacteria will grow and the media will turn a bright blue as a result of an increase in the pH of the media.

MOTILITY TEST

The motility test is not a biochemical test since we are not looking at metabolic properties of the bacteria. Rather, this test can be used to check for the ability of bacteria to migrate away from a line of inoculation. To perform this test, the bacterial sample is inoculated into motility media using a needle. Simply stab the media in as straight a line as possible and withdraw the needle very carefully to avoid destroying the straight line. After incubating the sample for 24-48 hours observations can be made. Check to see if the bacteria have migrated away from the original line of inoculation. If migration away from the line of inoculation is evident then you can conclude that the test organism is motile (positive test). Lack of migration away from the line of inoculation indicates a lack of motility (negative test result).

COAGULASE TEST

This test is used to identify Staphylococcus aureus which produces the enzyme coagulase, it causes plasma to clot by converting fibrinogen to fibrin. Two types of

coagulase are produced by most strains of S.aureus, free coagulase and bound coagulase. Free coagulase converts fibrinogen to fibrin by activating a coagulase reacting factor present in plasma. Free coagulase is detected by clotting in the tube test. Bound coagulase converts fibrinogen directly to fibrin without requiring a coagulase factor. It can be detected by the clumping of bacterial cells in the rapid slide test. A tube test must always be performed when the result of a slide test is not clear, or when the slide test is negative and Staphylococcus has been isolated from a serious infection.

STEP 4 - ANTIBIOTIC SENSITIVITY TEST

- After various test mentioned above will identify the genus and species of bacteria. Then the grown colonies of organism are spread over the Mueller Hinton Agar media plate. Labelled antibiotic discs are placed over the grown colonies of organism on the Mueller Hinton Agar media plate by the help of sterilized forceps. This plate will be again incubated for 12 hours to 24 hours at 37^{o}C. A zone of inhibition is appeared surrounding the antibiotic disc indicates the sensitivity of the organism to the particular antibiotic and the zone of inhibition is measured by the help of WHO quality control Chart to access the sensitivity of above mentioned discs.

FOLLOW UP

Clinical sign and symptoms were recorded frequently to assess the dissolution of infection.

PROCEDURE FOR ANAEROBIC CULTURE

STEP 1- SAMPLE COLLECTION

- After wearing sterilized gloves, site is anesthetized with depending on the condition of pus sample either collected intraorally or extra orally. The pus samples were collected by sterile 18 gauge and 38mm length needle with 5 ml syringe. After aspiration any free air was discharged and needle was capped immediately and material was dispensed in following transport medium. The transport medium used in the study was 1.5ml brain heart infusion broth which was sealed in a glass jar to maintain the anaerobic (condition) atmosphere in the tube for the survival of the anaerobic bacteria.

STEP 2 - TRANSPORTATION OF COLLECTED SAMPLE

- The commonly used transport media for storing the sample in viable condition from the site of collection of sample to the laboratory are:
- PBS – Phosphate buffered saline
- RS – Ringers Solution
- BHI – Brain Heart infusion Broth
- For the present study BHI was used as the transport media.
- The clinical sample is then transported to the laboratory immediately for anaerobic culture.

BRAIN HEART INFUSION BROTH

Composition

1. Proteose Peptone Mixture - 10.00 gm/litre
2. Disodium Phosphate - 2.50 gm/litre
3. Beef Heart Infusion - 10.00 gm/litre
4. Sodium Chloride - 5.00 gm/litre
5. Calf Brain Infusion - 7.50 gm/litre
6. Beef Heart Infusion - 10.00 gm/litre
7. Dextrose - 2.00 gm/litter

Preparation: Suspended 37 grams of the medium in 1000 ml of distilled water, and it is mixed well until complete dissolution of the medium occurs. The medium was sterilized in autoclave at 15 lbs pressure (121ºC) for 15 minutes. The prepared medium should be stored at 8 to15°C. The color of the preparation is clear amber and slightly opalescent. The nutritionally rich base of Beef heart and Calf brain infusions and Peptone mixture provides nitrogen, vitamins, minerals and amino acids that support the growth of a variety of microorganisms. Disodium phosphate acts as a buffer. Dextrose is the fermentable carbohydrate providing carbon and energy. Sodium chloride maintains the osmotic balance.

STEP 3- ANAEROBIC INCUBATION

- The sample is kept in the incubator without disturbing the anaerobic atmosphere for 24 hours. Turbidity is observed after 24 hours confirming the growth of the organism.

STEP 4 - SPECIMEN CULTURE

- Meanwhile the following media plates are prepared and kept for sterility check for 24 hours.

  (i) Nutrient agar

  (ii) Blood agar supplemented with hemin and vitamin

  (iii) MacConkey agar

  (iv) TSBV agar

NUTRIENT AGAR

Composition

Ingredients	**Gms / Litre**
Peptic digest of animal tissue	5.000
Sodium chloride	5.000
Beef extract	1.500
Yeast extract	1.500
Agar	15.000

Final pH (at 25°C) 7.4±0.2

Preparation- Add 28g of nutrient agar in 1000ml of distilled water. The mixture is then heat to boiling till the medium dissolve completely. Then the medium is sterilized by

Autoclaving at 15 lbs pressure (121°C) for 20 minutes. Allow this solution to cool to ~ 50°C. If leave it too long, it will solidify in the jar and will be of no use. Do not pour the solution when it is too hot as it may damage the petri dishes. Pour solution into petri dishes and allow it to cool to room temperature to set. Plates can be stored in the fridge for up to 4 weeks for use.

BLOOD AGAR SUPPLEMENTED WITH HEMIN AND VITAMIN

Composition

Per Liter Purified Water

Pancreatic Digest of Casein	-	10.0 g
Peptic Digest of Animal Tissue	-	10.0 g
Yeast Extract	-	2.0 g
Glucose	-	1.0 g
Sodium Chloride	-	5.0 g
Sodium Bisulfite	-	0.1 g
Hemin	-	0.005 g
Vitamin K1	-	0.01 g
Agar	-	15.0 g
Sheep Blood, defibrinated	-	5%

TRYPTIC SOY-SERUM IN BACITRACIN VANCOMYCIN AGAR (TSBV)

- TSBV is an enriched selective media for the isolation and presumptive identification of A.actinomycetemcomitans.
- TSVB contains Bacitracin and Vancomycin at a concentration that inhibits most gram-positive and gram negative anaerobes, except for A.actinomycetemcomitans.
- Storage: at room temperature in original container until use. Overheating and freezing is avoided.
- Shelf Life: 90 days from date of manufacture.

Composition

Tryptic Soy Agar	-	40.0 g
Yeast Extract	-	1.0 g
Bacitracin	-	75.0 mg
Vancomycin	-	5.0 mg
Horse Serum	-	100.0 ml
Distilled Water	-	1000.0 ml

Final pH 7.1 +/- 0.2 at 25 degrees C.

- After sterility check, the incubated clinical sample is streaked on to the prepared sterile media plates. Quadrant streaking is most preferred streaking method.

- The plates are then sealed with paraffin tape (adhesive tape) such that no air enters the plates.

- Anaerobic jar with gas pack and indicator tablets are kept ready.

STEP 5- ANAEROBIC INCUBATION

- The plates are immediately placed in the anaerobic jar packed tightly by the screw on top of the lid. Then the anaerobic jar is sealed using the paraffin tape.

- The anaerobic jar is then kept in the incubator, which is kept under 37^0 C for 5-7 days; undisturbed.

ANAEROBIC INCUBATOR

- Incubation of the streaked media plates is to be done under anaerobic conditions.

- For this purpose, anaerobic jar is used. It is a plastic transparent jar with a lid with a rubber tube for maintaining tight anaerobic condition.

- The jar is packed tight with the lid that is tightened with the help of a screw on the top of the lid.

- The anaerobic condition is maintained inside the jar by placing a Gas pack in it. Gas pack has two components, CO_2 releaser and a small porous pack called the O_2 absorber.

- An indicator tablet is placed inside the jar. The tablet is usually pink in colour but when exposed to anaerobic atmosphere, it turns blue in colour. When placed in the anaerobic jar and the sealed, the anaerobic condition is indicated by colour change of the indicator tablet to pink.

- Thus the anaerobic jar along with gas pack and indicator tablet forms the complete anaerobic atmosphere for the incubation of the clinical sample streaked on media plates.

STEP 6- IDENTIFICATION OF MICROBES

- After the usual incubation period of 5-7 days, the plates are taken out of the anaerobic jar. The plates are now ready for the observation of colonies and further biochemical and antibiogram tests. Results obtained are recorded and the isolated colonies are sub cultured for further reference.

BIOCHEMICAL TESTS

- For the characterization of the isolated colonies, biochemical tests are performed:

  - Indole Test

  - Citrate Test

- ❖ Urease Test
- ❖ Nitrate Test
- ❖ Triple Sugar Iron Test

➢ Results for the above biochemical tests were observed and recorded for characterization of isolated colonies.

STEP 7- ANTIBIOTIC SENSITIVITY TEST

After various test mentioned above we will take Mueller Hinton Agar, and the colonies of bacteria are spread over this medium. Discs are placed which are mentioned above by the help of sterilized forceps. This plate will be again incubated for 12 hours to 24 hours at 37^{o}C. Zone of Inhibition is measured by the help of WHO quality control Chart to access the sensitivity of above mentioned discs.

FOLLOW UP

Clinical signs and symptoms were recorded frequently to assess the dissolution of infection.

Results

STATISTICAL ANALYSIS

The data was analyzed by student t test and Chi square test for statistical significance within group and between groups. For the analysis SPSS (16.0 version) was used. All the data has been represented as frequencies and proportions. The data was evaluated using chi square test. $P<0.05$ is considered as statistically significant.

TABLE-1: DISTRIBUTION OF ODONTOGENIC INFECTION

In this study out of 142 patients, 125 cases organisms were isolated and taken up for the study, Out of 125 cases, 29 (23.2%) Aerobic organisms, 36 (28.8%) anaerobic organisms and 60 (48%) mixed organisms were isolated.

GRAPH-1: TOTAL NUMBER OF AEROBIC, ANAEROBIC AND MIXED INFECTION

*$P<0.05$ significant compared aerobics with anaerobic and mixed, $^{\#}P<0.05$ significant compared anaerobic with mixed. This frequency of infection between the organisms was statistically significant.

TABLE-2: TOTAL NUMBER OF INFECTIONS

In this study, a total of 10 sites were found to be affected. Out of 125 cases, 46cases were Vestibular Space followed by 24 Submandibular, 16 buccal, 10 canine, 8 submassetric, 6 canine with buccal, 5 sublingual, 4 palatal space, Temporal and Ludwigs were affected in 3 cases each. In these infections most common site was vestibular space

followed by other spaces. This frequency of infection between the spaces was statistically significant.

GRAPH-2: COMPARISON OF FREQUENCY AND PERCENTAGE OF TOTAL MICRO-ORGANISM ISOLATED

125 cases organisms were isolated and taken up for the study, Out of 125 cases, 29 (23.2%) Aerobic organisms, 36 (28.8%) anaerobic organisms and 60 (48%) mixed organisms were isolated.

P<0.05 significant compared aerobics with anaerobic and mixed, P<0.05 significant compared anaerobic with mixed. This frequency of infection between the organisms was statistically significant.

TABLE-3: NUMBER, TYPE, FREQUENCY AND PERCENTAGE OF AEROBIC MICRO-ORGANISMS ISOLATED

GRAPH-3: NUMBER AND PERCENTAGE OF AEROBIC ORGANISMS

Table-3 and Graph-3 shows out of 29 aerobic organisms 10 (34.49%) were *Streptococcus viridans* and was the most common organism isolated followed by *6* (20.69%) *Staphylococcus aureus*, 5 (17.25 %) Coagulase negative *Staphylococcus*, 4 (13.79 %) *Pseudomonas aeruginosa* and both *Escherichia coli* and *Klebsiella Pneumonia* were isolated from 2 (6.89 %) specimens each.

P<0.05 significant compared *Streptococcus viridians* with other organisms,

TABLE-4: NUMBER, TYPE, FREQUENCY AND PERCENTAGE OF ANAEROBIC MICRO-ORGANISMS ISOLATED

GRPAH-4: NUMBER AND PERCENTAGE OF ANAEROBIC ORGANISMS

Table-4 and Graph-4 shows anaerobic group of 36 organisms, 22 (61.11%) *Peptostreptococcus* and was the most common organism isolated followed by10 (27.78%) *Bacteroides*, and 4 (11.11 %) *Actinomyces*.

P<0.05 significant compared *Peptostreptococcus* with other organisms.

TABLE-5: NUMBER, TYPE, FREQUENCY AND PERCENTAGE OF MIXED MICRO-ORGANISMS ISOLATED

GRAPH-5: NUMBER AND PERCENTAGE OF MIXED MICRO-ORGANISM ISOLATED

Table-5 and Graph-5 shows the number, type, frequency and percentage of mixed micro-organisms isolated. *Streptococcus viridians with Peptostreptococcus* were isolated from 18 (30%) cases and was the most common mixed organism isolated followed by *Staphylococcus aureus with Peptostreptococcus* in 12 (20%) cases, *Streptococcus Viridans with Bacteroides* in 9 (15 %) cases, *Staphylococcus Aureus with Bacteroides* in 6 (10 %) cases, *Coagulase negative staphylococcus with Bacteroides* from 1 (1.67 %) cases, *Streptococcus Viridans with Actinomyces* in 8 (13.33 %) cases and *Staphylococcus Aureus with Actinomyces* in 6 (10 %) cases. P<0.05 significant compared *Streptococcus viridians & Peptostreptococcus* with other organisms.

TABLE-6: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO SULFONAMIDES AND FLUROQUINOLONES

GRAPH-6 AND GRAPH-7: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO SULFONAMIDES AND FLUROQUINOLONES

Table-6, Graph-6 and Graph-7 shows, sensitivity to Ofloxacin was 17 (58.62%) out of 29 aerobic organisms, 19 (52.77%) out of 36 anaerobes and 25 (41.66%) out of 60 mixed organisms. Resistance to Ofloxacin was 12 (14.36%) out of 29 aerobic organisms, 17 (47.22%) out of 36 anaerobic organisms and 35 (58.33%) out of 60 mixed organisms. Sensitivity to Levofloxacin was 12 (41.37%) out of 29 aerobes, 20 (55.55%) out of 36 anaerobes and 31 (51.67%) out of 60 mixed organisms. Resistance to Levofloxacin was 17 (58.62%) out of 29 aerobes, 16 (44.44%) out of 36 anaerobes and 29 (48.33%) out of 60 mixed infections, followed by Gatifloxacin and norfloxacin and ciprofloxacin. Organisms showed least sensitivity and higher resistance towards Cotrimoxazole and sulfadiazine.

$P<0.05$ significant compared Ofloxacin with other drugs.

TABLE-7: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO QUINOLONES GROUP

Among Qunolones, all the aerobic, anaerobic and mixed organisms were resistant to Nalidixic acid.

TABLE-8: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO AMINOGLYCOSIDE GROUP.

GRAPH-8 AND GRAPH 9: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO AMINOGLYCOSIDE GROUP.

Table-8, Graph-8 and Graph-9 shows, among the Aminoglycoside group, out of 29 aerobic organisms 5 (17.24%) were sensitive and 24 (82.76%) were resistant, out of 36 cases of anaerobic organisms 3 (8.33%) were sensitive and 33 (91.67%) were resistant and for mixed organisms 12 (12%) were sensitive and 48 (80.00%) were resistant to Gentamycin followed by Amikacin and Neomycin. Streptomycin showed least sensitive and highest resistant.

$P<0.05$ significant compared Gentamycin with other drugs in the group.

TABLE-9: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO MACROLIDE GROUP.

GRAPH 10 AND GRAPH 11: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO MACROLIDE GROUP.

Table-9, Graph-10 and Graph-11 shows, among the Macrolide group organisms were least sensitive to Erythromycin (3.45%) and higher resistant (96.67%). But sensitivity to Azitromycin was 6 (20.69%) out of 29 aerobic organisms, 8(22.22%) out of 36 anaerobic organisms, and 18 (30%) out of mixed organisms. Resistance to Azitromycin was 23 (79.31%) out of 29 aerobic organisms, 28 (77.78%) out of 36 anaerobic organisms and 42 (70%) out of 60 mixed organisms followed by Roxithromycin.

P<0.05 significant compared Azitromycin with other drugs in the group.

TABLE-10: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO PENICILLIN GROUP.

GRAPH 12 AND GRAPH 13: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO PENICILLIN GROUP

Table-10, Graph-12 and Graph-13 shows, among the pencillins group there was resistance to Amoxicillin in 28 (96.55%) out of 29 aerobic organisms, 31 (86.11%) out of 36 anaerobic organisms and 58 (86.33%) of mixed organisms, followed by ampicillin, cloxacillin, pencillin, augmentin (amoxicillin with clavulenic acid) showed the highest resistance. On the other hand sensitivity to Piperacillin was 19 (65.52%) out of 29 aerobic organisms, 20 (55.56%) out of 36 anaerobic organisms and, 35(58.33%) out of 60 mixed organisms. Resistance to Piperacillin was 10 (27.78%) out of 29 aerobic organisms and 16 (44.44%) out of 36 anaerobic organisms and 25 (41.67%) out of 60 mixed organisms.

P<0.05 significant compared Piperacillin with other drugs in the group.

TABLE-11: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO CEPHALOSPORIN GROUP.

GRAPH 14 AND GRAPH 15 : NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO CEPHALOSPORIN GROUP.

Table-11, Graph-14 and Graph-15 shows, In Cephalosporins group, Sensitivity to Cefixime was 20 (68.97%) out of 29 aerobic organisms, 28 (77.78%) out of 36 anaerobic

organisms and 45 (75%) out of 60 mixed organisms. Resistance to Cefixime was 9 (31.03%) out of 29 aerobic organisms, 8 (22.22%) out of 36 anaerobic organisms and 15 (25%) out of 60 mixed organisms. Sensitivity to Cefotaxime was 18 (62.07%) out of 29 aerobic organisms, 24 (66.67%) out of 36 anaerobic organisms and 39 (65%) out of 60 mixed organisms. Resistance to Cefotaxime was 11 (37.93%) out of 29 aerobic organisms, 12 (33.33%) out of 36 anaerobes and 21 (35%) out of 60 mixed organisms. Followed by ceftriaxone, cephalexin, cefradiazone, and the least sensitive in the group were cephpodoxime and cefazolin.

P<0.05 significant compared Cefixime with other drugs in the group.

TABLE-12: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO BROAD SPECTRUM ANTIBIOTICS.

GRAPH 16 AND GRAPH 17: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO BROAD SPECTRUM ANTIBIOTICS

Table-12, Graph-16 and Graph-17 shows, among the board spectrum antibiotics, organisms showed highest sensitivity and least resistance to Doxycyclin. Sensitivity to Doxycyclin was 15 (51.72%) out of 29 aerobes, 21 (58.33%) out of 36 anaerobic organisms and 34 (56.67%) out of mixed organisms. Resistance to doxycyclin was 14 (48.27%) out of 29 aerobic organisms, 15 (41.67%) out of 36 anaerobic organisms and 26 (43.33%) out of 60 mixed organisms. There was highest resistance and least sensitivity towards Tetracycline, Chloramphenicol and Meropenem.

P<0.05 significant compared Doxycyclin with other drugs in the group.

TABLE-13: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO MISCELLANEOUS GROUP OF DRUGS.

GRAPH 18 AND GRAPH 19: NUMBER AND PERCENTAGE OF CASES SENSITIVE AND RESISTANT TO MISCELLANEOUS GROUP OF DRUGS

Table-13, Graph-18 and Graph-19 shows, Among the miscellaneous group of drugs, all the aerobic, anaerobic and mixed group of organisms was sensitive to Linezolid (100%). Entire anaerobic group were sensitive (100%) to Metronidazole and 39 (65%) out of 60 mixed organisms were sensitive to metronidazole. Resistance to metronidazole was 21 (35%) out of 60 mixed organisms. Entire aerobic group were Sensitive (100%) to Clindamycin, 30 (83.33%) out of 36 anaerobic groups were sensitive to clindamycin. And 38 (63.33%) out of 60 mixed organisms were sensitive to clindamycin. Resistance to clindamycin was 6 (16.77%) out of 36 anaerobes. and 22 (36.67%) out of 60 mixed organisms. Vancomycin and bacitracin showed highly resistant and least sensitive. Furoxone, Nitrofurantoin, Septran, Sporidex showed highly resistant.

$P<0.05$ significant compared Linezolid with other drugs in the group.

TABLE-14,15,&16: ANTIBIOTICS WHICH ARE SENSITIVE AND RESISTANT AGAINST AEROBIC, ANAEROBIC AND MIXED ORGANISMS

Table number 14,15 and 16 shows the antibiotics which are sensitive and resistant to aerobic, anaerobic and mixed group of organisms.

Discussion

Orofacial infections are caused by variety of bacteria. In vitro culture and antibiotic sensitivity testing should be routinely performed for proper antibiotic selection. Indiscriminate use and easy availability of antibiotics without prescription has probably contributed to the development of bacterial resistance. Presence of these refractory bacterial strains increases the importance of culture and sensitivity test to ensure that an effective antibiotic is prescribed that causes resolution of the infection. Present study conducted on a local population with head and neck fascial space infection helped to identify the causative microbiota and their sensitivity and resistance to commonly used antibiotics.

Most odontogenic infections arise as a sequel to dental caries and periodontal infection. Most of the odontogenic infections resolve with little consequences although occasionally complications may lead to more severe infection of head and neck, particularly in immuno-compromised or debilitated patients[14]. In the present study most of the source of odontogenic infections was grossly carious tooth, periodontal infection followed by pericoronitis.

Oral infections spread in a pathway of least resistance often into the oral cavity or into the deep spaces of the neck, which may become life threatening. Deep-space infections originate most commonly from odontogenic sources in adults and from tonsil and other lymphatic sources in children. Spaces in the neck are created between the superficial, middle and deep layers of the deep cervical fascia. These spaces are interconnected[15].

In the literature Submandibular space is the most commonly involved in multiple space infection followed by lateral pharyngeal, buccal and submental[11, 23]. This was supported by Opeyemi O. Daramola and Poeschl PW.[23, 24]. In the present study of 125 cases we also found Submandibular space (20%) to be the most commonly involved in multiple space infection. In our study Out of the single space infections, Vestibular Space 46 (36.8%)showed more predisposition, deviating from literature by A.J Raga[10] et al who said the Submandibular space was the most common location for a single-space abscess (30.0%). M. Sakaguchi (1997)[21] reported peritonsillar space as the most common site. Thomas R Flynn (2006)[22] reported highest occurrence of infection in the pterygomaxillary space.

In the present study a total of 10 sites were found to be affected. Out of 125 cases, 46 (36.8%) cases were Vestibular Space and were the followed by 24 (20%) Submandibular, 16 (12.8%) buccal, 10 (10%) canine, 8 (6.4%) submassetric, 6 (4.8%) canine and buccal, 5 (4%) sublingual, 4 (3.2%) palatal space, Temporal and Ludwigs were affected in 3 (2.4%) cases each. In these infections most common site was vestibular space and submandibular space followed by other spaces.

The typical odontogenic infection is caused by a mixture of aerobic and anaerobic bacteria, approximately70% of these infections are caused by mixed flora. Pure aerobic bacteria are less common accounting for only 5%. Similarly pure anaerobic infections are approximately 25% of odontogenic infections[25].

The present study also supports the similar results, In 125 cases, organisms were isolated. Out of 125 cases, 29 (23.2%) were aerobic organisms, 36 (28.8%) were anaerobic organisms and in 60 (48%) cases mixed organisms were isolated. This result also shows that the head and neck space infections of odontogenic origin are poly microbial in nature. According to the literature the most of the head and neck infections of odontogenic origin are of polymicrobial in nature[1, 8, 9, 10, 11, 12, 13, 14, 15, 16].

In the present study most of the infections are of mixed organisms 60(48%), followed by pure anaerobes in 36(28%) and pure aerobes in 29(23.3%) cases. Andrew Bridgeman[13] in 1995 states that Odontogenic maxillofacial infections consist of aerobic, facultative anaerobic and obligate anaerobic bacteria with the aerobes and facultative anaerobes being outnumbered by strict anaerobic bacteria by a factor of at least 2:l with *streptococci* predominate. Timothy M. Osborn[26] in 2008 also reports that anaerobic bacteria are found to be more in odontogenic infections. J. E. Turner[33], et al in 1975 reported that aerobes predominate in odontogenic infections.

In this study, out of 125 cases, pure aerobic organisms caused infection in 29 cases. Out of this, 10 (34.49%) were *Streptococcus viridians* followed by *Staphylococcus aureus 6* (20.69%) cases, *Coagulase negative Staphylococcus* 5 (17.25 %) cases, *Pseudomonas Aeruginosa* 4 (13.79 %) cases, and both *Escherichia coli* and *Klebsiella Pneumonia* were isolated from 2 (6.89 %) specimens each.

In our study anaerobic group, out of 36 cases, 22 (61.11%) were *Peptostreptococcus* followed by*10* (27.78%) *Bacteroides*, and 4 (11.11 %) *Actinomyces*. Mixed species found were *Streptococcus viridians with Peptostreptococcus* isolated from 18 (30%) cases and was the most common mixed organism isolated followed by *Staphylococcus aureus with Peptostreptococcus* in 12 (20%) cases, *Streptococcus Viridans with Bacteroides* in 9 (15 %) cases, *Staphylococcus Aureus with Bacteroides* in 6 (10 %) cases, *Coagulase negative staphylococcus with Bacteroides* in 1 (1.67 %) cases, *Streptococcus Viridans with Actinomyces* in 8 (13.33 %) cases and *Staphylococcus Aureus with Actinomyces* in 6 (10 %) cases.

According to the literature it has been found that *Streptococus viridians* was the most common pathogens in the head and neck space infections[1,10, 19,21,26, 27, 28, 29, 30,33,51] and in our study *Streptococus viridians* was also found to be the most common microorganism. The second most common microorganism isolated was the *Staphylococcus* aureus 5 (17.25 %). These results were also supported by previous studies[7, 34, 35].

The third most common micro organism isolated in this study was *Coagulase negative Staphylococcus* 4 (13.79 %) which was found an important source of nosocomial infections. Methicillin resistant *Coagulase negative Staphylococci* is associated with prophylactic and therapeutic use of cephalosporins (Laith Hussein *et al.* (2010)[36]. In our study, we found significant level of resistance to commonly used antibiotics in (6%) *Coagulase negative Staphylococci*.

The two most popular theories for the origin of antibiotic resistance are. (1) Resistance may be either inherits or (2) it may be acquired by the processes of genetic mutation or gene transfer.[17, 18]

Mechanism of acquired resistance falls into one of the five categories, although bacteria may employ more than one mechanism[17, 18, 36]

1. Alteration of drugs target site

2. Inability of a drug to reach the target site

3. Inactivation of an antimicrobial agent

4. Active elimination of an antibiotic from the cell

Specific mechanisms by which bacteria acquire resistance genes are[17]

1. Spontaneous mutation

2. Gene transfer

3. Bacteriophages

4. Mosaic genes

Therefore proper selection of antibiotics has a twofold benefit: (1) the rapid elimination of infection, which decreases the extent of tissue destruction; and (2) diminishing the use of improper antibiotics to prevent the development of antibiotic

resistance. In addition to becoming familiar with the indigenous microbiota of oral cavity, the patient's immune status, allergy profile, and previous antibiotic usage that may predispose to resistant organisms need to be considered[15]. The antibiotics chosen by the surgeon must be effective against microbes that are most likely to cause abscess in orofacial region and it should decrease the development of bacterial resistance. Usually most of the odontogenic infections are successfully managed by incision and drainage, together with extraction or root canal therapy and proper antibiotic regime[37, 38, 39]. However sometimes infections from these spaces may spread into other danger spaces leading to life threatening conditions. Timely and deliberate efforts to establish debridement and drainage as well as appropriate antibiotic therapy should be initiated by the surgeon.[4]

The laboratory data regarding microbiological flora, antibiotic susceptibility are crucial information for the clinician who is considering the administration of antibiotic therapy. However it may take several days to obtain such data. Hence antibiotics are chosen empirically.

Penicillin has been the antibiotic of choice for most odontogenic infections[12,13,14,34,37]. But resistant organisms have developed due to its long and widespread use[40, 41, 42, 43]. Although penicillin remains the antibiotic of choice for mild to moderate odontogenic infections in the immunocompetent host but pencillin should not be used as initial therapy for more serious infections possibly involving penicillin resistant oral anaerobes[45].

In our center Amoxicillin with Metronodazole were the most common empirical antibiotics prescribed to patients with odontogenic infections. And in our study among the pencillins group, there was resistance to Amoxicillin in 28 (96.55%) out of 29 aerobic organisms, 31 (86.11%) out of 36 anaerobic organisms and 58 (86.33%) of mixed organisms, followed by Ampicillin, Cloxacillin, Pencillin, Augmentin (amoxicillin with clavulenic acid) showed the highest resistance. On the other hand sensitivity to Piperacillin was 19 (65.52%) out of 29 aerobic organisms, 20 (55.56%) out of 36 anaerobic organisms and, 35(58.33%) out of 60 mixed organisms. Resistance to Piperacillin was 10 (27.78%) out of 29 aerobic organisms and 16 (44.44%) out of 36 anaerobic organisms and 25 (41.67%) out of 60 mixed organisms.

In this study among the Macrolide group, organisms were least sensitive to Erythromycin (3.45%) and higher resistance (96.67%). But sensitivity to Azitromycin was 6 (20.69%) out of 29 aerobic organisms, 8(22.22%) out of 36 anaerobic organisms, and 18 (30%) out of mixed organisms. Resistance to Azitromycin was 23 (79.31%) out of 29 aerobic organisms, 28 (77.78%) out of 36 anaerobic organisms and 42 (70%) out of 60 mixed organisms followed by Roxithromycin. This high resistance to macrolides was supported by Paul. W. Poeschl *et al.* in 2010[48], he concluded that the high resistance rate for macrolides was especially striking and may necessitate an adoption of newer antibiotic regime in the future.

In our study among the fluroqunolones, organisms showed highest sensitivity towards Ofloxacin and Levofloxacin. Sensitivity to Ofloxacin was 17 (58.62%) out of 29

aerobic organisms, 19 (52.77%) out of 36 anaerobes and 25 (41.66%) out of 60 mixed organisms. Resistance to Ofloxacin was 12 (14.36%) out of 29 aerobic organisms, 17 (47.22%) out of 36 anaerobic organisms and 35 (58.33%) out of 60 mixed organisms. Sensitivity to Levofloxacin was 12 (41.37%) out of 29 aerobes, 20(55.55%) out of 36 anaerobes and 31(51.67%) out of 60 mixed organisms. Resistance to Levofloxacin was 17 (58.62%) out of 29 aerobes, 16 (44.44%) out of 36 anaerobes and 29 (48.33%) out of 60 mixed infections, followed by Gatifloxacin and norfloxacin and ciprofloxacin. Organisms showed least sensitivity and higher resistance towards Cotrimoxazole and sulfadiazine. This result was supported by Munish Kohli[47] et al in 2009 in his study Ofloxacin was the most sensitive drug. The most resistant drugs were amoxicillin and ampicillin. The gram negative colonies were sensitive to cefotaxime.

In our study, among aminoglycosides 5 (17.24%) out of 29 aerobic organisms, 3 (8.33%) out of 36 anaerobic organisms and 12 (12%) out of 60 mixed organisms were sensitive to Gentamycin. Resistance to Gentamycin was noted in 24 (82.76%) out of 29 aerobes, 33 (91.67%) out of 36 anaerobes, and 48 (80.00%) out of 60 mixed organisms, Streptomycin showed least sensitive and highest resistant.

In the present study among Cephalosporins group, Sensitivity to Cefixime was 20 (68.97%) out of 29 aerobic organisms, 28 (77.78%) out of 36 anaerobic organisms and 45 (75%) out of 60 mixed organisms. Resistance to Cefixime was 9 (31.03%) out of 29 aerobic organisms, 8 (22.22%) out of 36 anaerobic organisms and 15 (25%) out of 60 mixed organisms. Sensitivity to Cefotaxime was 18 (62.07%) out of 29 aerobic organisms, 24 (66.67%) out of 36 anaerobic organisms and 39 (65%) out of 60 mixed organisms. Resistance to Cefotaxime was 11 (37.93%) out of 29 aerobic organisms, 12

(33.33%) out of 36 anaerobes and 21 (35%) out of 60 mixed organisms. Followed by ceftriaxone, cephalexin, cefradiazone, and the least sensitive in the group were cephpodoxime and cefazolin.

Among the board spectrum antibiotics, organisms showed highest sensitivity and least resistance to Doxycyclin. Sensitivity to Doxycyclin was 15 (51.72%) out of 29 aerobes, 21 (58.33%) out of 36 anaerobic organisms and 34 (56.67%) out of mixed organisms. Resistance to doxycyclin was 14 (48.27%) out of 29 aerobic organisms, 15 (41.67%) out of 36 anaerobic organisms and 26 (43.33%) out of 60 mixed organisms. There was highest resistance and least sensitivity towards Tetracycline, Chloramphenicol and Meropenem.

Among the miscellaneous group of drugs, all the aerobic, anaerobic and mixed group of organisms was sensitive to Linezolid (100%). Entire anaerobic group were sensitive (100%) to Metronidazole and 39 (65%) out of 60 mixed organisms were sensitive to metronidazole. Resistance to metronidazole was 21 (35%) out of 60 mixed organisms. Entire aerobic group were Sensitive (100%) to Clindamycin, 30 (83.33%) out of 36 anaerobic groups were sensitive to clindamycin. And 38 (63.33%) out of 60 mixed organisms were sensitive to clindamycin. Resistance to clindamycin was 6 (16.77%) out of 36 anaerobes and 22 (36.67%) out of 60 mixed organisms. Vancomycin and bacitracin showed high resistance and least sensitivity. Furoxone, Nitrofurantoin, Septran, Sporidex showed high resistance to all the group of drugs. John G. Bartlett[49] et al in 1975 reported that Clindamycin proved equally effective in anaerobic pulmonary infections and in his study there were no therapeutic failures reported with clindamycin.

Vejayan Krishnan, in 1993[4] described that pencillin resistant organisms have developed due to its long and widespread use so that Clindamycin became preferred antibiotic for empiric therapy in his study. In literature, lots of reports about the resistance of Amoxycillin and alternative antibiotic should be replaced such as Clindamycin [8, 21,22]. Even clindamycin failure with penicillin therapy and a rate of penicillin resistance also has been reported.

Specificity of empirical antibiotic therapy could be improved with good knowledge about the pathologic flora in the locality. This will help in administration of appropriate antibiotics instantaneously to control the infection. This will counter the delay in identification of the causative agent and specific antibiotic therapy.

Summary& Conclusion

This study was aimed at identifying the major causative aerobic and anaerobic micro organisms involved in head and neck fascial space infections of odontogenic origin and their sensitivity and resistance to commonly used antibiotics.

The study showed most of the head and neck fascial space infections are of odontogenic origin and are polymicrobial in nature, majority being mixed organisms and more than one half are pure anaerobes. The most common aerobic organism isolated was *Streptococcus viridians,* most common anaerobe was *Peptostreptococci,* and the most common mixed organism was *Streptococcus with Peptostreptococci*.

Amoxicillin was the most commonly used empirical antibiotic in all the cases and showed highest resistance for all organisms. But all the aerobic, anaerobic and mixed group of organisms was sensitive to Linezolid. The entire anaerobic group showed sensitive to Metronidazole. The entire aerobic group showed sensitive to Clindamycin. Among Fluroquinolones organisms showed highest sensitivity towards Ofloxacin, Levofloxacin and Cefixime and Cefotaxime among Cephalosporins group.

According to the study there should be substitution of miscellaneous group of antibiotics such as Linezolid, Clindamycin, third generation cephalosporins such as

cefixime, cefotaxime, and fluroqunolones such as Ofloxacin and Levofloxacin for Amoxicillin in the empirical management of deep fascial space infections. Hence successful management of head and neck fascial space infections of odontogenic origin can be achieved by appropriate surgical intervention to establish drainage, good overall supportive care of the patient, gram staining of purulent exudates to provide immediate information needed for the rational selection of an antibiotic and *in vitro* microbiological culture and antibiotic susceptibility.

It can be concluded that the knowledge about the pathologic flora involved in head and neck infection in a locality and their sensitivity and resistance to commonly used antibiotics will help the clinician in administering appropriate antibiotics at the earliest phase of infection, which will adequately control the infection and hence minimizing the morbidity.

Tables

Table-1: Distribution of Odontogenic Infection

S. No	Place	Aerobic		Anaerobic		Mixed	
		Number	Percentage	Number	Percentage	Number	Percentage
1	Vestibular Space	10	34.48	14	38.44	22	36.67
2	Sub Mandibular Space	6	20.68	8	22.22	10	16.66
3	Buccal Space	3	10.34	4	11.11	9	15.00
4	Canine Space	2	6.90	3	8.33	5	8.33
5	Sub Massetric Space	2	6.90	2	5.55	4	6.67
6	Canine and Buccal Space	2	6.90	1	2.78	3	5.00
7	Sub lingual Space	1	3.45	1	2.78	3	5.00
8	Palatal Space	1	3.45	1	2.78	2	3.33
9	Ludwigs Angina	1	3.45	1	2.78	1	1.67
10	Temporal Space	1	3.45	1	2.78	1	1.67
	Total	**29**	**100**	**36**	**100**	**60**	**100**

Table-2: Total number of Infections

S. No	Site	Total number
1	Vestibular Space	46
2	Sub Mandibular Space	24
3	Buccal Space	16
4	Canine Space	10
5	Sub Massetric Space	8
6	Canine and Buccal Space	6
7	Sub lingual Space	5
8	Palatal Space	4
9	Ludwigs Angina	3
10	Temporal Space	3
	Total	**125**

Table-3: Number, type, frequency and percentage of aerobic micro-organisms isolated

S. No.	Aerobic Organisms	Frequency	Percentage (%)
1	Streptococcus Viridans	10	34.49
2	Staphylococcus Aureus	6	20.69
3	Coagulase negative staphylococcus	5	17.25
4	Pseudomonas Aeroginosa	4	13.79
5	E. Coil	2	6.89
6	Klebsiella Pneumonia	2	6.89
	Total	**29**	**100**

Table-4: Number, type, frequency and percentage of Anaerobic micro-organisms isolated

Sl. No	Anaerobic Organisms	Frequency	Percentage (%)
1	Peptostreptococcus	22	61.11
2	Bacteroides	10	27.78
3	Actinomyces	4	11.11
	Total	**36**	**100**

Table-5: Number, type, frequency and percentage of mixed micro-organisms isolated

S. No	Mixed Organism	Frequency	Percentage (%)
1	Streptococcus Viridans + Peptostreptococcus	18	30.00
2	Staphylococcus Aureus + Peptostreptococcus	12	20.00
3	Streptococcus Viridans + Bacteroides	9	15.00
4	Staphylococcus Aureus + Bacteroides	6	10.00
5	Coagulase negative staphylococcus + Bacteroides	1	1.67
6	Streptococcus Viridans + Actinomyces	8	13.33
7	Staphylococcus Aureus + Actinomyces	6	10.00
	Total	**60**	**100**

Table-6: Number and percentage of cases sensitive and resistant to Sulfonamides and Fluroquinolones

Drug	Aerobic				Anaerobic				Mixed			
	Sensitive		Resistant		Sensitive		Resistant		Sensitive		Resistant	
	No	%	No	%	No	%	No	%	No	%	No	%
Ofloxacin	17	58.62	12	14.36	19	52.77	17	47.22	25	41.66	35	58.33
Ciprofloxacin	2	6.89	27	93.10	0	0	36	100	3	5.00	57	95.00
Cotrimoxazole	0	0	29	100	0	0	36	100	0	0	60	100
Norfloxacin	3	10.34	26	89.65	2	5.55	34	94.44	7	11.67	53	88.33
Sulfadiazine	0	0	29	100	0	0	36	100	1	1.67	59	98.33
Gatifloxacin	6	20.68	23	79.31	3	8.33	33	91.67	9	15.00	51	85.00
Levofloxacin	12	41.37	17	58.62	20	55.55	16	44.44	31	51.67	29	48.33

Table-7: Number and percentage of cases sensitive and resistant to Quinolone group

Drug	Aerobic				Anaerobic				Mixed			
	Sensitive		Resistant		Sensitive		Resistant		Sensitive		Resistant	
	No	%	No	%	No	%	No	%	No	%	No	%
Nalidixic acid	0	0	29	100	0	0	36	100	0	0	60	100

Table-8: Number and percentage of cases sensitive and resistant to Aminoglycoside group

Drug	Aerobic				Anaerobic				Mixed			
	Sensitive		Resistant		Sensitive		Resistant		Sensitive		Resistant	
	No	%	No	%	No	%	No	%	No	%	No	%
Amikacin	4	13.79	25	86.21	7	19.44	29	80.55	10	16.67	50	83.33
Gentamycin	5	17.24	24	82.76	3	8.33	33	91.67	12	20.00	48	80.00
Neomycin	1	3.45	28	96.55	2	5.55	34	94.44	5	8.33	55	91.67
Streptomycin	0	0	29	100	0	0	36	100	0	0	60	100

Table-9: Number and percentage of cases sensitive and resistant to Macrolide group

Drug	Aerobic				Anaerobic				Mixed			
	Sensitive		Resistant		Sensitive		Resistant		Sensitive		Resistant	
	No	%	No	%	No	%	No	%	No	%	No	%
Erythromycin	1	3.45	28	96.55	0	0	36	100	2	3.33	58	96.67
Azithromycin	6	20.69	23	79.31	8	22.22	28	77.78	18	30.00	42	70.00
Roxithromycin	4	13.79	25	86.21	5	13.89	31	86.11	10	16.67	50	83.33

Table-10: Number and percentage of cases sensitive and resistant to Penicillin group

Drug	Aerobic				Anaerobic				Mixed			
	Sensitive		Resistant		Sensitive		Resistant		Sensitive		Resistant	
	No	%	No	%	No	%	No	%	No	%	No	%
Amoxicillin	1	3.44	28	96.55	5	13.89	31	86.11	7	11.67	53	88.33
Ampicillin	1	3.44	28	96.55	1	2.78	35	97.22	3	5.00	57	95.00
Piperacillin	19	65.52	10	27.78	20	55.56	16	44.44	35	58.33	25	41.67
Cloxacillin	1	3.44	28	96.55	3	8.33	33	91.67	3	5.00	57	95.00
Penicillin	1	3.44	28	96.55	0	0	36	100	0	0	60	100
Augmentin	2	6.89	27	93.10	0	0	36	100	1	1.67	59	98.33

Table-11: Number and percentage of cases sensitive and resistant to Cephalosporin group

Drug	Aerobic				Anaerobic				Mixed			
	Sensitive		Resistant		Sensitive		Resistant		Sensitive		Resistant	
	No	%	No	%	No	%	No	%	No	%	No	%
Cefotaxime	18	62.07	11	37.93	24	66.67	12	33.33	39	65.00	21	35.00
Cefixime	20	68.97	9	31.03	28	77.78	8	22.22	45	75.00	15	25.00
Cephalexin	3	10.34	26	89.66	1	2.78	35	97.22	5	8.33	55	91.67
Cefpodoxime	1	3.45	28	96.55	1	2.78	35	97.22	3	5.00	57	95.00
Cefradiazone	1	3.45	28	96.55	3	8.33	33	91.67	3	5.00	57	95.00
Cefazolin	1	3.45	28	96.55	1	2.78	35	97.22	3	5.00	57	95.00
Ceftriaxone	1	3.45	28	96.55	5	13.89	31	86.11	5	8.33	55	91.67

Table-12: Number and percentage of cases sensitive and resistant to Broad spectrum antibiotics

Drug	Aerobic				Anaerobic				Mixed			
	Sensitive		Resistant		Sensitive		Resistant		Sensitive		Resistant	
	No	%	No	%	No	%	No	%	No	%	No	%
Chloramphenicol	1	3.45	28	77.78	0	0	36	100	2	3.33	58	96.67
Doxycycline	15	51.72	14	48.27	21	58.33	15	41.67	34	56.67	26	43.33
Meropenem	1	3.45	27	75.00	2	5.56	34	94.44	5	8.33	55	91.67
Tetracycline	1	3.45	27	75.00	1	2.78	35	97.22	4	6.67	56	93.33

Table-13: Number and percentage of cases sensitive and resistant to miscellaneous group of drugs

Drug	Aerobic				Anaerobic				Mixed			
	Sensitive		Resistant		Sensitive		Resistant		Sensitive		Resistant	
	No	%	No	%	No	%	No	%	No	%	No	%
Linezolid	29	100	0	0	36	100	0	0	60	100	0	0
Metronidazole	0	0	29	100	36	100	0	0	39	65.00	21	35.00
Vancomycin	1	3.45	28	96.55	1	2.78	35	97.22	3	5.00	57	95.00
Bacitracin	1	3.45	28	96.55	10	27.78	26	72.22	20	33.33	40	66.67
Clindamycin	29	100	0	0	30	83.33	6	16.67	38	63.33	22	36.67
Furoxone	0	0	29	100	0	0	36	100	0	0	60	100
Nitrofurantoin	0	0	29	100	0	0	36	100	0	0	60	100
Septran	0	0	29	100	0	0	36	100	0	0	60	100
Sporidex	0	0	29	100	0	0	36	100	0	0	60	100

Table-14: Antibiotics which are sensitive and resistant agonist aerobic organisms.

S. No	Microorganisms	Sensitive	Resistant
1.	**Streptococcus Viridans**	Ofloxacin, Norfloxacin Gatifloxacin, Levofloxacin, Gentamicin, Azithromycin, Roxithromycin, Piperacillin, Cloxacillin, Meropenem, Cefotaxime, Cefixime, Cefuroxime, Cefpodoxime, Doxycycline, Clindamycin.	Sulfadiazine, Nalidixic acid, Ciprofloxacin, Cotrimoxazole, Amikacin, Neomycin, Streptomycin, Erythromycin, Ampicillin, Amoxicillin, Penicillin, Augmentin Cephalexin, Ceftazidime, Cefazolin, Tetracycline, Chloramphenicol, Bacitracin, Vancomycin, Furoxone, Nitrofurantoin, Septran, Sporidex.
2.	**Staphylococcus Aureus**	Gatifloxacin, Norfloxacin, Ofloxacin, Levofloxacin, Gentamicin, Azithromycin, Roxithromycin, Cloxacillin, Cefotaxime, Cefixime, Cefuroxime, Cefpodoxime, Doxycycline, Linezolid, Clindamycin.	Sulfadiazine, Nalidixic acid, Ciprofloxacin, Cotrimoxazole, Amikacin, Neomycin, Streptomycin, Erythromycin, Ampicillin, Amoxicillin, Penicillin, Piperacillin, Augmentin, Cephalein, Ceftazidime, Cefazolin, Tetracycline, Meropenem, Chloramphenicol, Bacitracin, Vancomycin, Furoxone, Nitrofurantoin, Septran, Sporidex.
3.	**Pseudomonas Aeruginosa**	Ofloxacin, Gentamicin, Amikacin, Roxithromycin, Piperacillin, Cefotaxime, Cefixime Cefradiazone, Levofloxacin, Doxycycline, Linezolid, Clindamycin.	Sulfadiazine, Nalidixic acid, Gatifloxacin Ciprofloxacin, Norfloxacin Cotrimoxazole, Neomycin, Streptomycin, Erythromycin Azithromycin, Ampicillin, Amoxicillin, Penicillin, Augmentin, Cloxacillin, Cephalein, Cefpodoxime, Cefazolin, Cefuroxime, Tetracycline, Chloramphenicol, Meropenem, Bacitracin, Vancomycin, Furoxone, Nitrofurantoin, Septran, Sporidex.

4.	**E. coil**	Ofloxacin, Norfloxacin, Gentamicin, Amikacin, Roxithromycin, Azithromycin, Piperacillin, Cloxacillin, Cefotaxime, Cefpodoxime, Cefradiazone, Cefixime, Levofloxacin, Doxycycline, Clindamycin, Linezolid.	Sulfadiazine, Nalidixic acid Gatifloxacin Cotrimoxazole, Ciprofloxacin, Neomycin, Streptomycin, Erythromycin, Ampicillin, Amoxicillin, Penicillin, Augmentin, Cephalein, Cloxacillin, Cefazolin Cefuroxime, Tetracycline, Chloramphenicol, Meropenem, Bacitracin, Vancomycin, , Furoxone, Nitrofurantoin, Septran, Sporidex.
5.	**Klebsiella Pneumonia**	Ofloxacin, Ciprofloxacin, Gentamicin, Cefotaxime, Cefixime, Cefradiazone, Levofloxacin, Linezolid, Clindamycin, Doxycycline.	Sulfadiazine, Nalidixic acid Norfloxacin, Gatifloxacin, Cotrimoxazole, Amikacin, Neomycin, Streptomycin, Erythromycin Azithromycin, Roxithromycin, Ampicillin, Amoxicillin, Penicillin, Augmentin Cloxacillin Piperacillin, Cephalein, Cefotaxime, Cefazolin, Cefuroxime, Tetracycline, Chloramphenicol, Meropenem, Bacitracin, Vancomycin, Furoxone, Nitrofurantoin, Septran, Sporidex.
6.	**Coagulase negative staphylococcus**	Ciprofloxacin, Gentamicin, Neomycin, Cefotaxime, Cefixime Cefradiazone, Cefpodoxime, Doxycycline, Linezolid, Clindamycin.	Sulfadiazine, Nalidixic acid, Ofloxacin, Norfloxacin, Gatifloxacin Cotrimoxazole, Amikacin, Streptomycin, Erythromycin Azithromycin, Roxithromycin, Ampicillin, Amoxicillin, Penicillin, Augmentin Cloxacillin, Cephalein, Cefazolin Cefuroxime, Tetracycline, Levofloxacin, Chloramphenicol, Meropenem, Bacitracin, Vancomycin, Furoxone, Nitrofurantoin, Septran, Sporidex.

Table-15: Antibiotics which are sensitive and resistant agonist anaerobic organisms.

S. No	Microorganisms	Sensitive	Resistant
1.	**Peptostreptococcus**	Gatifloxacin, Gentamicin, Amikacin, Azithromycin, Roxithromycin, Cloxacillin, Cefotaxime, Cefixime, Levofloxacin, Linezolid, Bacitracin, Metronidazole, Clindamycin.	Sulfadiazine, Nalidixic acid Ciprofloxacin, Ofloxacin, Norfloxacin, Cotrimoxazole, Neomycin, Streptomycin, Erythromycin, Ampicillin, Amoxicillin, Penicillin, Augmentin Piperacillin, Cephalein, Cefradiazone, Cefazolin, Cefuroxime, Cefpodoxime, Tetracycline, Meropenem Doxycycline, Chloramphenicol, Vancomycin, Furoxone, Nitrofurantoin, Septran, Sporidex.
2.	**Bacteroides**	Azithromycin, Bacitracin, Roxithromycin, Amoxicillin, Ceftriaxone, Cefotaxime, Cefixime, Levofloxacin, Meropenem, Linezolid, Metronidazole, Clindamycin.	Sulfadiazine, Nalidixic acid Ofloxacin, Ciprofloxacin, Sporidex, Cotrimoxazole Norfloxacin, Gatifloxacin, Amikacin, Neomycin, Streptomycin, Gentamicin, Septran Erythromycin, Cloxacillin, Ampicillin, Penicillin, Piperacillin, Augmentin, Cephalein, Cefradiazone, Cefazolin, Cefuroxime, Furoxene Cefpodoxime, Tetracycline, Doxycycline, Chloramphenicol, Vancomycin, Nitrofurantoin.
3.	**Actinomyces**	Ofloxacin, Ciprofloxacin, Gentamicin, Amikacin, Neomycin, Roxithromycin, Azithromycin, Piperacillin, Amoxicillin, Cefotaxime, Cefradiazone, Levofloxacin, Bacitracin, Linezolid, Metronidazole.	Sulfadiazine, Nalidixic acid, Norfloxacin, Gatifloxacin, Sooridex, Cotrimoxazole, Streptomycin Erythromycin, Ampicillin, Penicillin, Augmentin Cloxacillin, Cephalein, Cefpodoxime, Cefazolin Cefuroxime, Cefixime, Tetracycline, Doxycycline, Chloramphenicol, Meropenem, Vancomycin, Clindamycin, Furoxone, Nitrofurantoin, Septran.

Table-16: Antibiotics which are sensitive and resistant against mixed organisms.

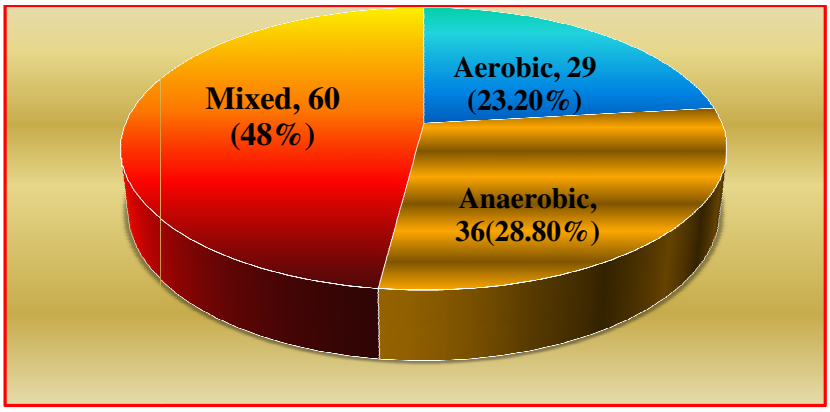
S. No	Mixed infection	Sensitive	Resistant
1.	**Streptococcus Viridans + Peptostreptococcus**	Ofloxacin, Norfloxacin, Gatifloxacin, Gentamicin, Amikacin, Roxithromycin, Azithromycin, Piperacillin, Cloxacillin, Cefotaxime, Cefixime Cefpodoxime, Cefuroxime, Levofloxacin, Doxycycline, Meropenem, Bacitracin, Clindamycin, Linezolid, Metronidazole.	Sulfadiazine, Nalidixic acid Cotrimoxazole, Ciprofloxacin, Streptomycin, Neomycin, Erythromycin, Amoxicillin, Ampicillin, Penicillin, Augmentin, Cephalein, Cefazolin Cefradiazone, Tetracycline, Chloramphenicol, Vancomycin, Furoxone, Nitrofurantoin, Septran, Sporidex.
2.	**Staphylococcus Aureus + Peptostreptococcus**	Sulfadiazine, Norfloxacin, Gatifloxacin, Gentamicin, Amikacin, Roxithromycin, Azithromycin, Cloxacillin, Cefotaxime, Cefixime, Cefpodoxime, Cefuroxime, Levofloxacin, Meropenem, Bacitracin, Linezolid, Metronidazole.	Nalidixic acid, Ofloxacin, Cotrimoxazole, Ciprofloxacin, Streptomycin, Neomycin, Erythromycin, Amoxicillin, Ampicillin, Piperacillin, Penicillin, Augmentin, Cephalein, Cefazolin, Cefradiazone, Tetracycline, Doxycycline, Chloramphenicol, Vancomycin, Clindamycin, Furoxone, Nitrofurantoin, Septran, Sporidex.
3.	**Streptococcus Viridans + Bacteroides**	Norfloxacin, Ofloxacin, Gatifloxacin, Gentamicin, Roxithromycin, Liezolid, Azithromycin, Cloxacillin, Amoxicillin, Piperacillin, Cefotaxime , Cefpodoxime, Cefuroxime, Levofloxacin, Meropenem, Doxycycline, Bacitracin, Metronidazole.	Sulfadiazine, Amikacin, Streptomycin, Neomycin, Erythromycin, Ampicillin, Penicillin, Augmentin, Cefixime, Cephalein, Cefazolin Cefradiazone, Tetracycline, Chloramphenicol, Vancomycin, Clindamycin, Furoxone, Nitrofurantoin, Septran, Sporidex.
4.	**Staphylococcus Aureus + Bacteroides**	Norfloxacin, Gatifloxacin, Roxithromycin, Gentamicin, Cloxacillin, Amoxicillin, Cefotaxime, Cefixime, Cefpodoxime, Cefuroxime, Levofloxacin, Meropenem, Bacitracin,	Sulfadiazine, Nalidixic acid Cotrimoxazole Ofloxacin,, Ciprofloxacin, Amikacin, Streptomycin, Neomycin, Erythromycin, Azithromycin, Piperacillin, Ampicillin, Penicillin, Augmentin, Cephalein,

		Linezolid, Metronidazole.	Cefazolin Cefradiazone, Tetracycline, Chloramphenicol, Doxycycline, Vancomycin, Clindamycin, Furoxone, Nitrofurantoin, Septran, Sporidex.
5.	**Coagulase negative staphylococcus + Bacteroides**	Ciprofloxacin, Gentamicin, Azithromycin, Cloxacillin, Piperacillin, Bacitracin, Linezolid, Metronidazole, Levofloxacin, Meropenem, Cefotaxime, Cefuroxime, Levofloxacin, Meropenem, Bacitracin, Linezolid, Metronidazole.	Sulfadiazine, Nalidixic acid Norfloxacin, Ofloxacin, Cotrimoxazole, Gatifloxacin, Amikacin, Streptomycin, Neomycin, Roxithromycin, Erythromycin, Tetracycline, Doxycycline, Chloramphenicol, Cefpodoxime, Cephalein, Cefixime, Cefazolin, Cefradiazone, Ampicillin, Penicillin, Augmentin, Amoxicillin, Cefpodoxime, Cephalein, Cefixime, Cefazolin Cefradiazone, Tetracycline, Doxycycline, Chloramphenicol, Vancomycin, Clindamycin, Furoxone, Nitrofurantoin, Septran, Sporidex.
6.	**Streptococcus Viridans + Actinomyces**	Sulfadiazine, Norfloxacin, Ofloxacin, Gatifloxacin, Gentamicin, Neomycin, Roxithromycin, Azithromycin, Cloxacillin, Piperacillin, Cefotaxime, Cefixime, Cefpodoxime, Cefradiazone, Levofloxacin, Doxycycline, Bacitracin, Linezolid, Metronidazole.	Nalidixic acid, Cotrimoxazole, Ciprofloxacin, Erythromycin, Amoxicillin, Ampicillin, Penicillin, Augmentin, Cephalein, Cefazolin, Cefuroxime, Tetracycline, Meropenem, Chloramphenicol, Vancomycin, Clindamycin, Furoxone, Nitrofurantoin, Septran, Sporidex.
7.	**Staphylococcus Aureus + Actinomyces**	Norfloxacin, Ofloxacin, Gatifloxacin, Ciprofloxacin, Gentamicin, Neomycin, Roxithromycin, Cloxacillin, Piperacillin, Cefotaxime, Cefixime, Cefuroxime, Levofloxacin, Doxycycline, Bacitracin,	Sulfadiazine, Nalidixic acid, Cotrimoxazole, Amikacin, Streptomycin, Azithromycin, Erythromycin, Amoxicillin, Ampicillin, Penicillin, Augmentin, Cephalein, Cefpodoxime, Cefazolin Cefradiazone, Tetracycline,

		Linezolid, Metronidazole.	Meropenem, Chloramphenicol, Vancomycin, Clindamycin, Furoxone, Nitrofurantoin, Septran, Sporidex.

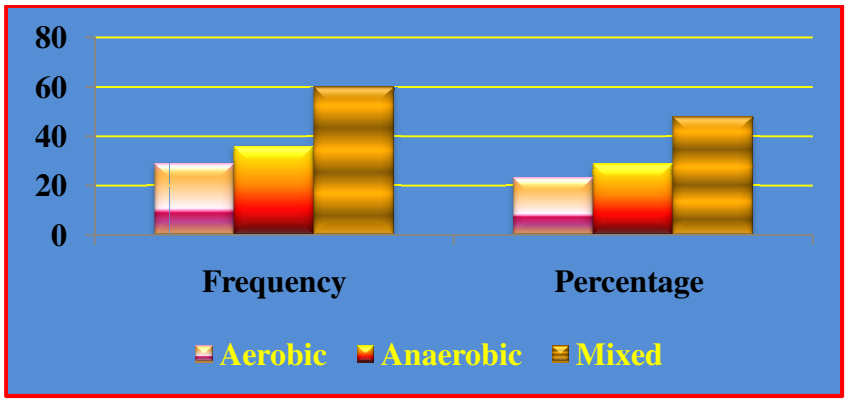
Graphs

Graph-1: Total number of aerobic, anaerobic and mixed infection

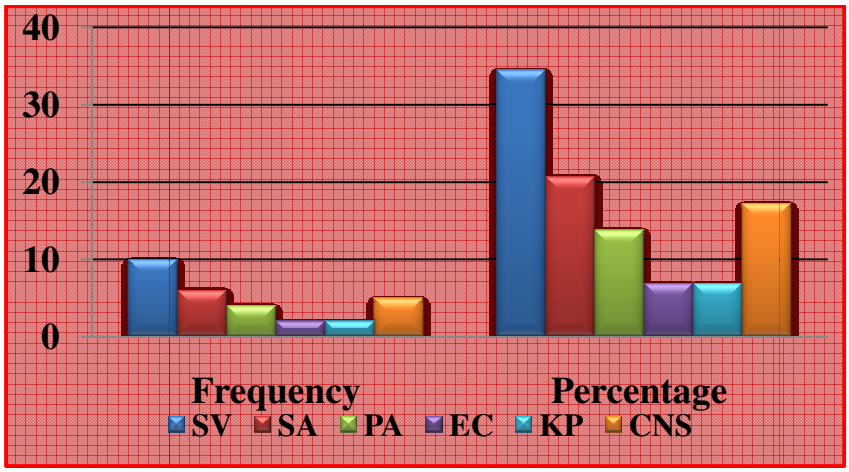


(*P<0.05 significant compared within the groups)

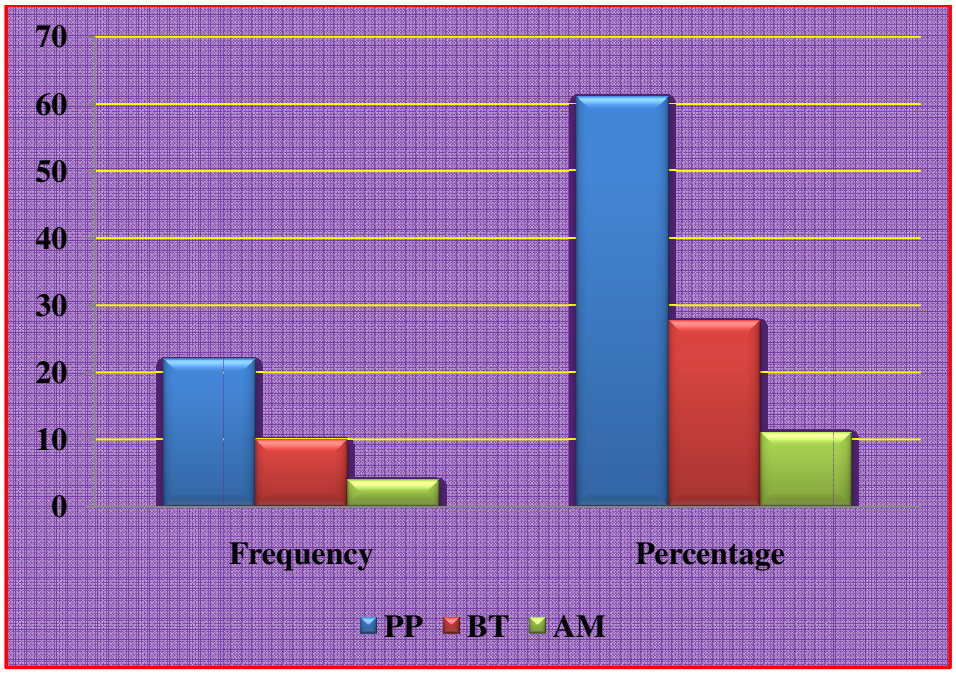
Graph-2: Comparison of frequency and percentage of total micro-organisms isolated



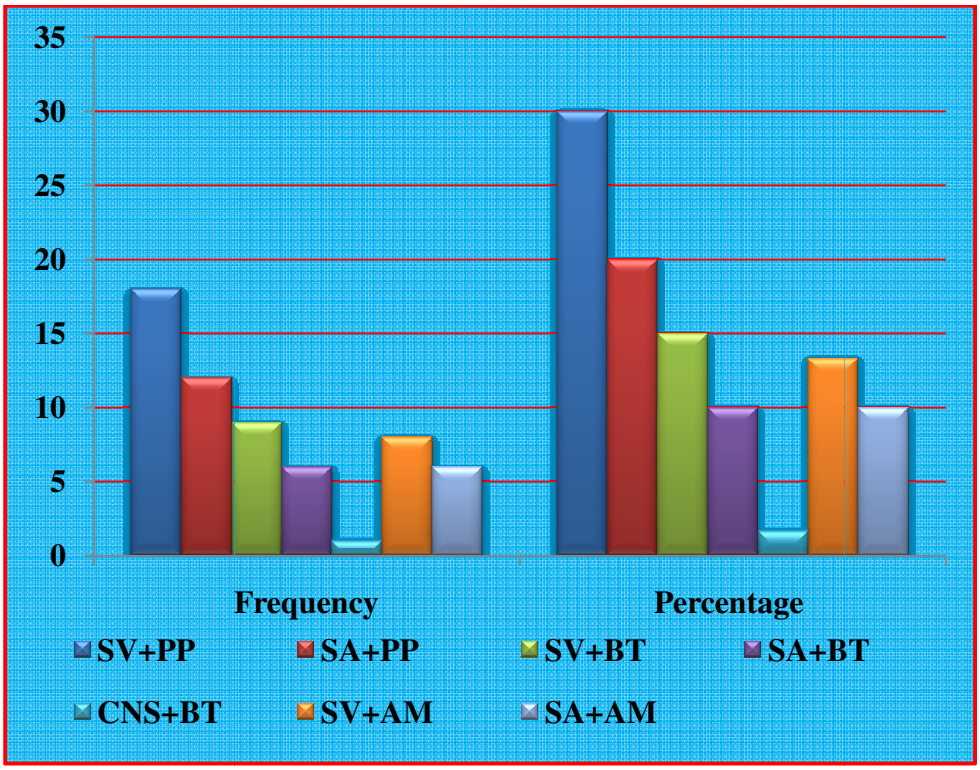
Graph-3: Number and percentage of aerobic organisms



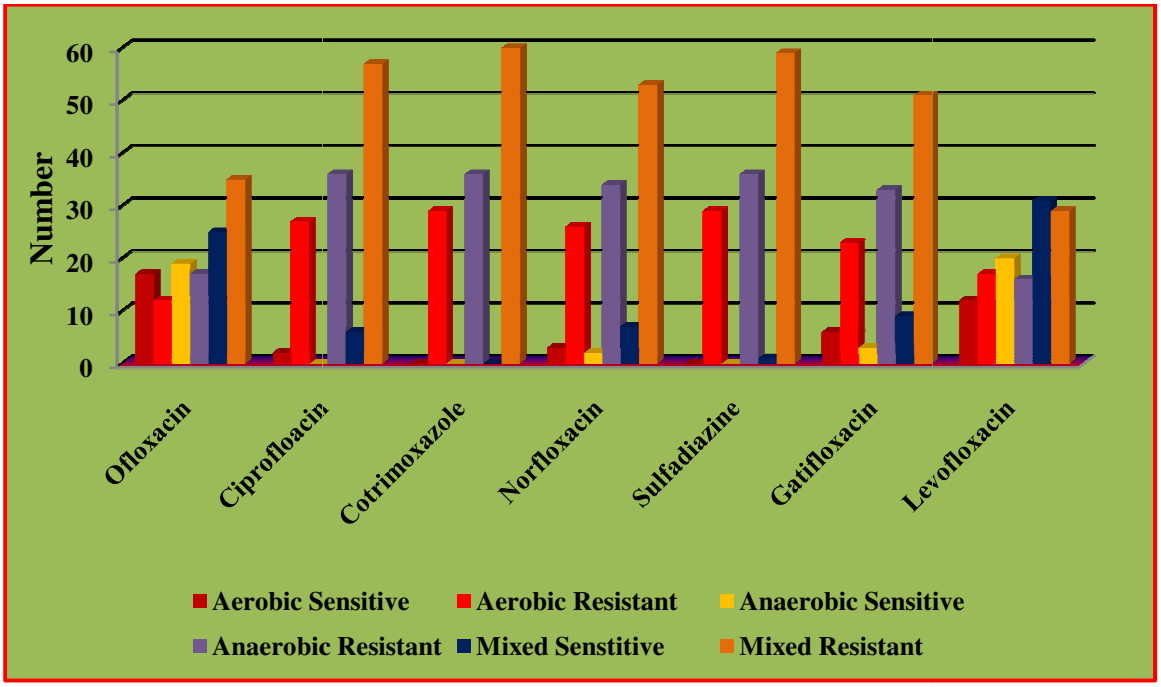
Grpah-4: Number and percentage of anaerobic organisms



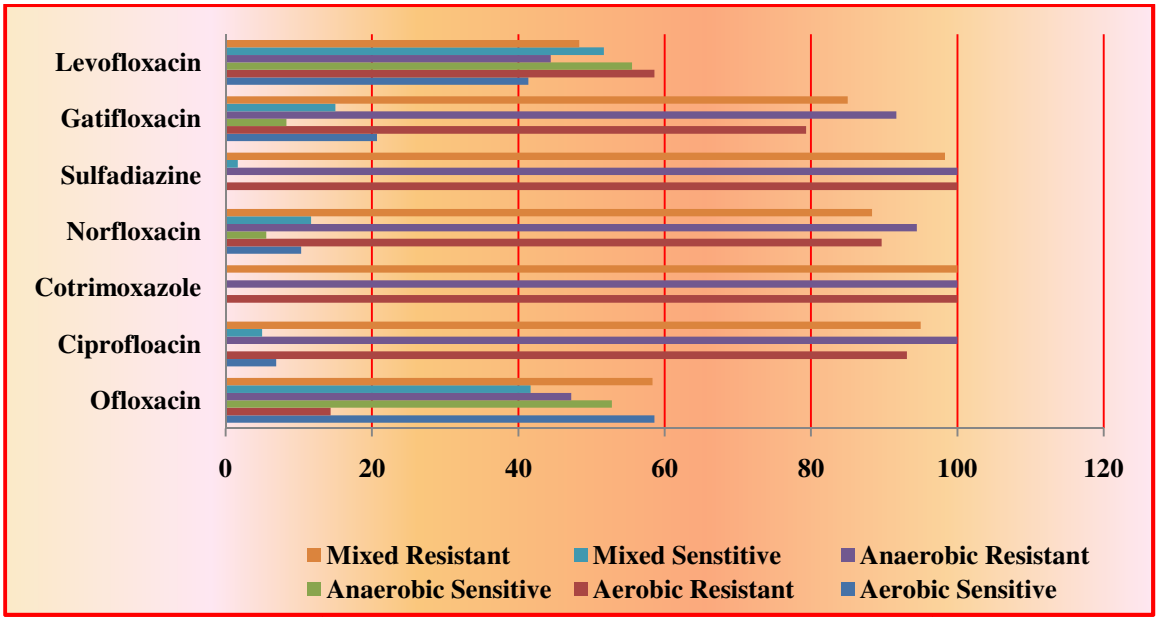
Graph-5: Number and percentage of mixed micro-organisms isolated



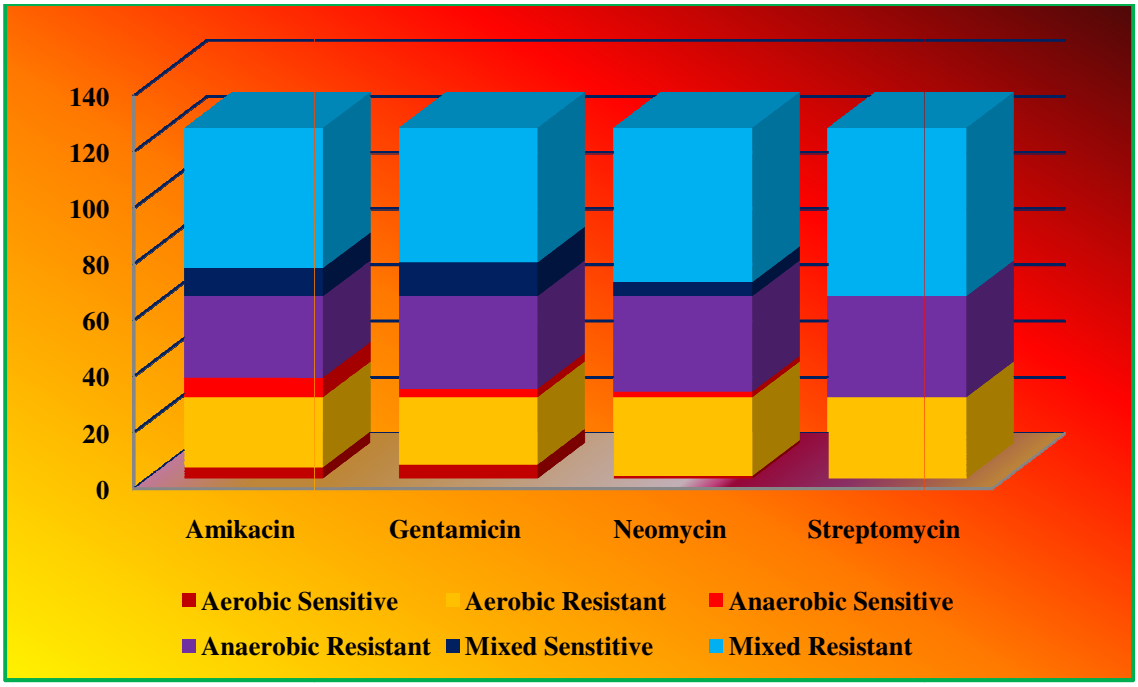
Graph-6: Number of cases sensitive and resistant to Sulfonamides and Fluroquinolones



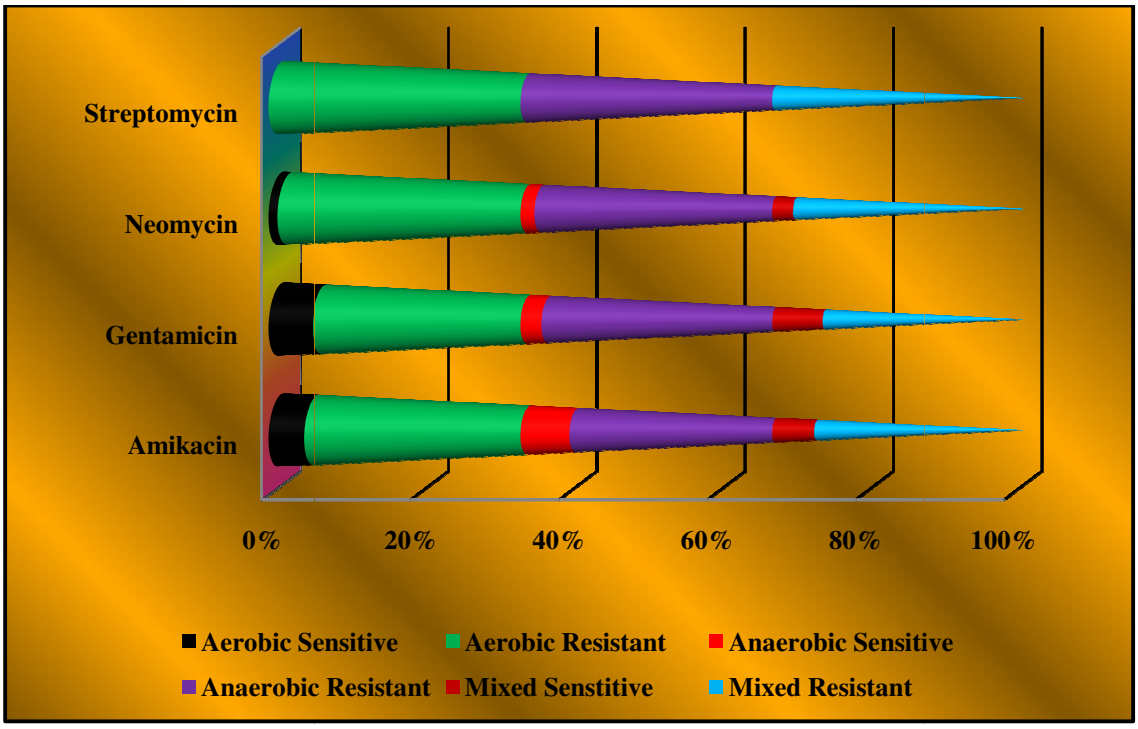
Graph-7: Percentage of cases sensitive and resistant to sulfonamides and Fluroquinolones



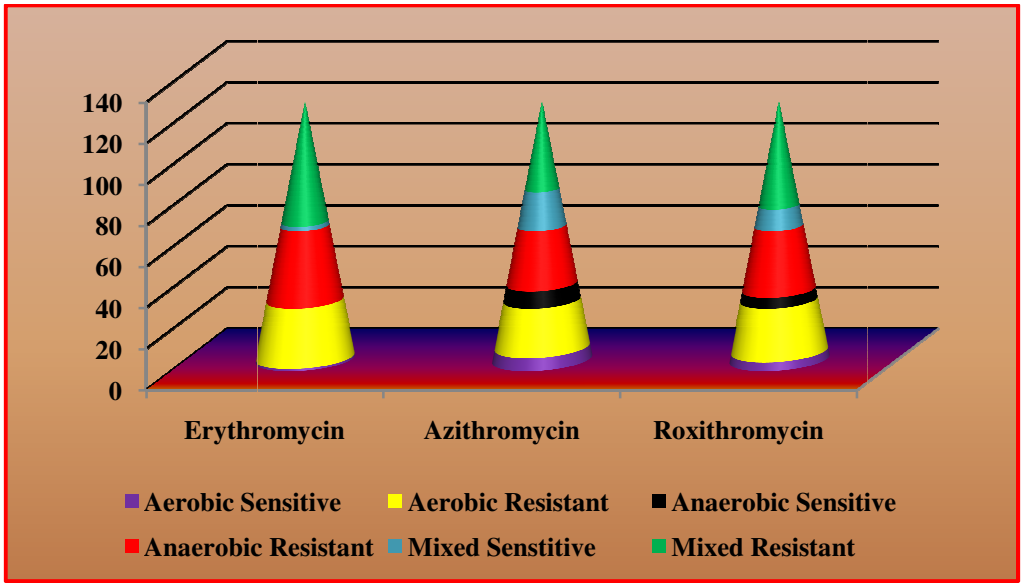
Graph-8: Number of cases sensitive and resistant to Aminoglycoside group



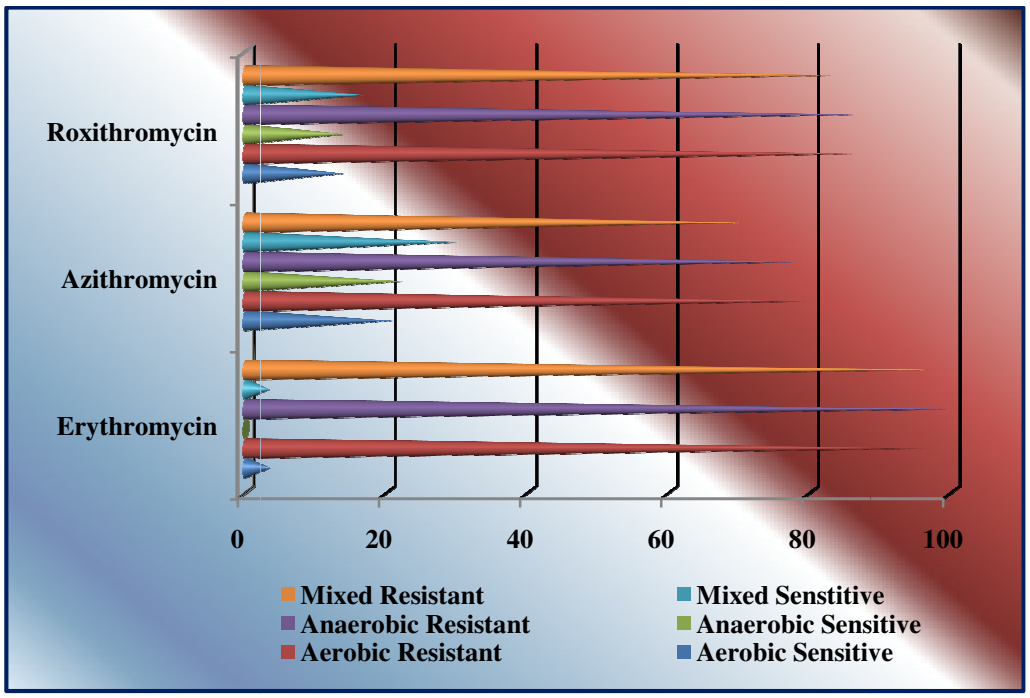
Graph 9: Percentage of cases sensitive and resistant to Aminoglycoside group



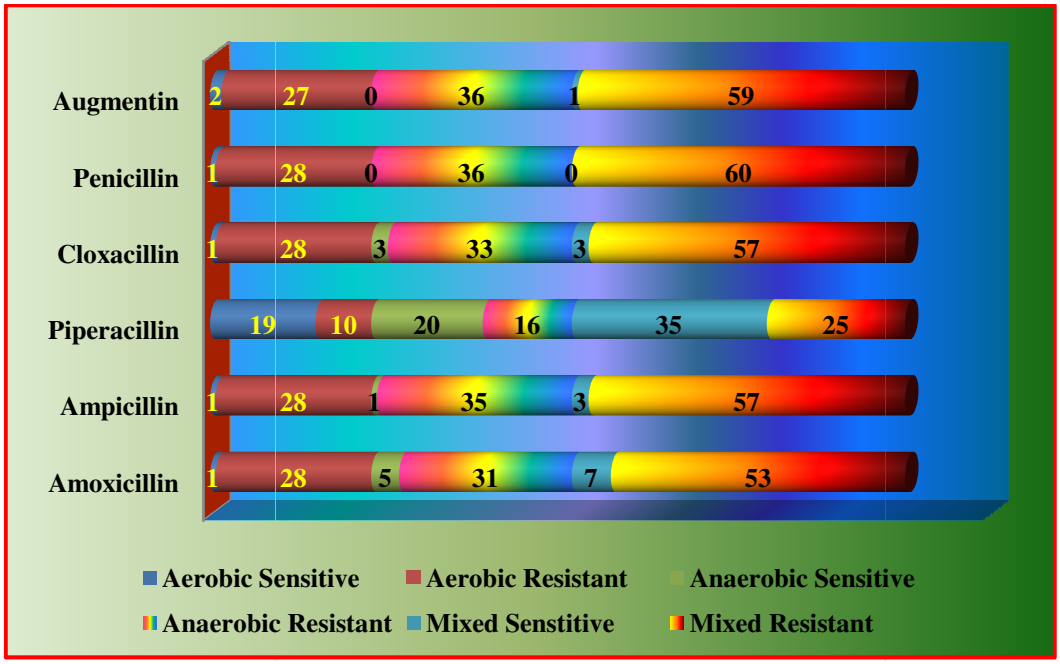
Graph 10: Number of cases sensitive and resistant to Macrolide group



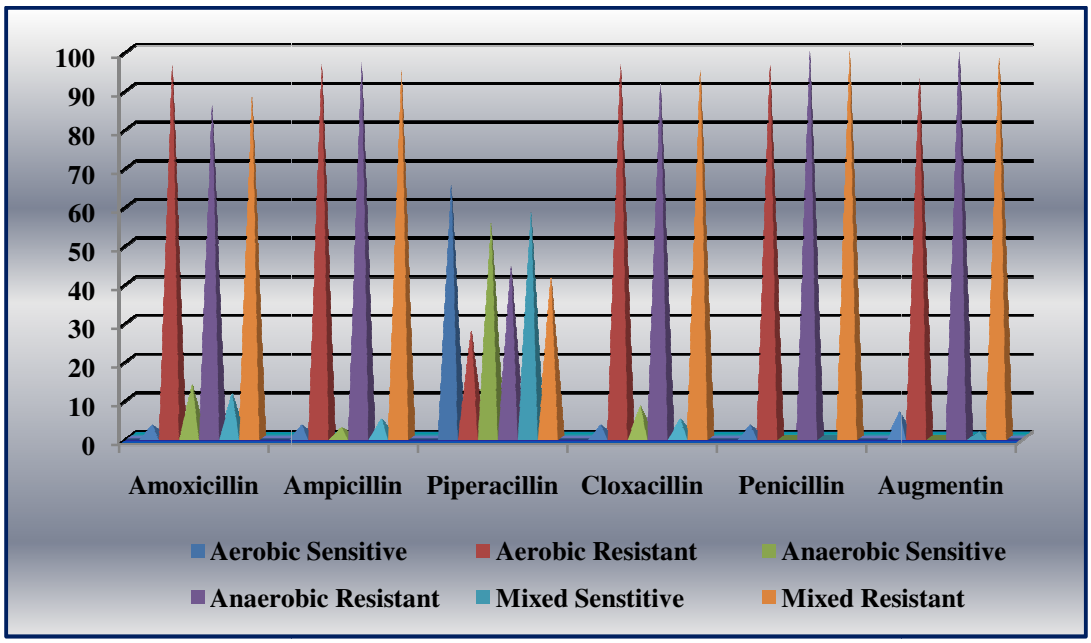
Graph 11: Percentage of cases sensitive and resistant to Macrolide group



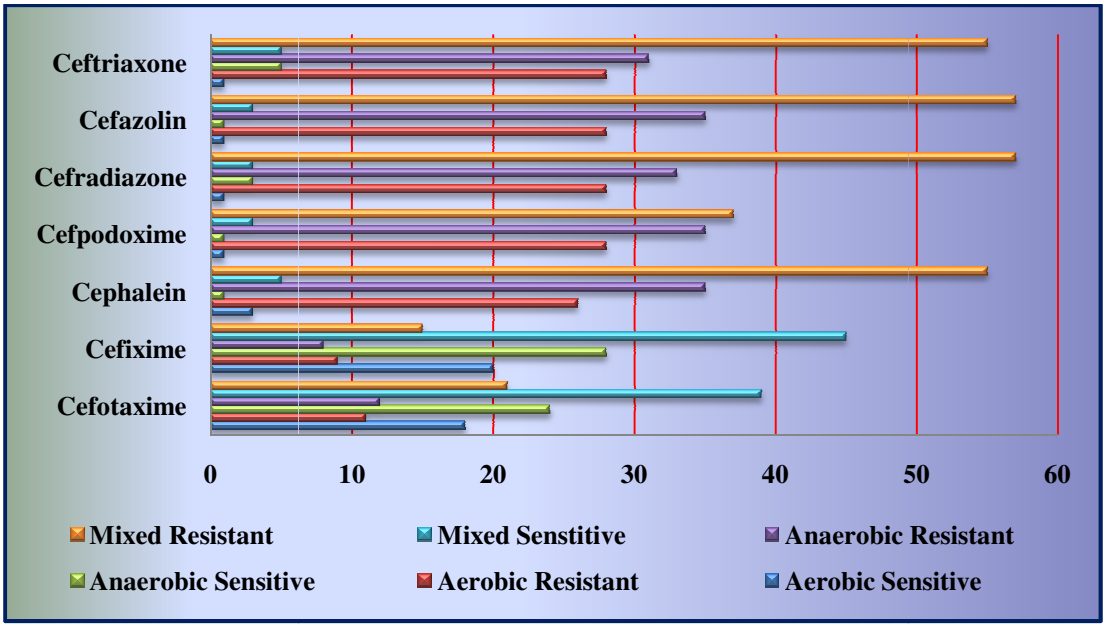
Graph 12: Number of cases sensitive and resistant to Penicillin group



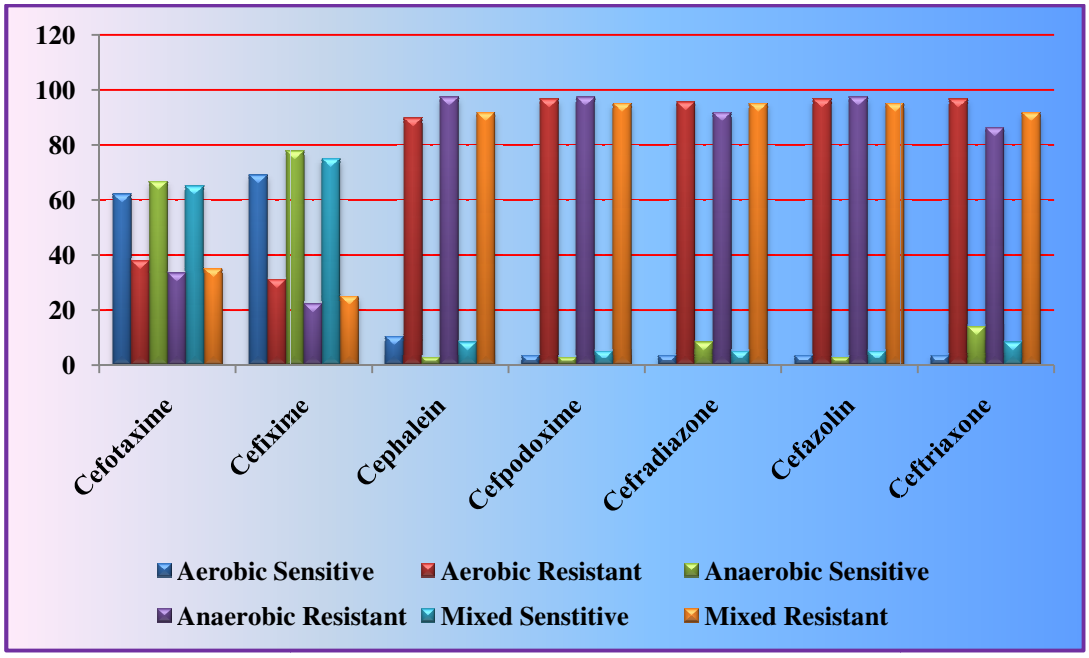
Graph 13: Percentage of cases sensitive and resistant to Penicillin group



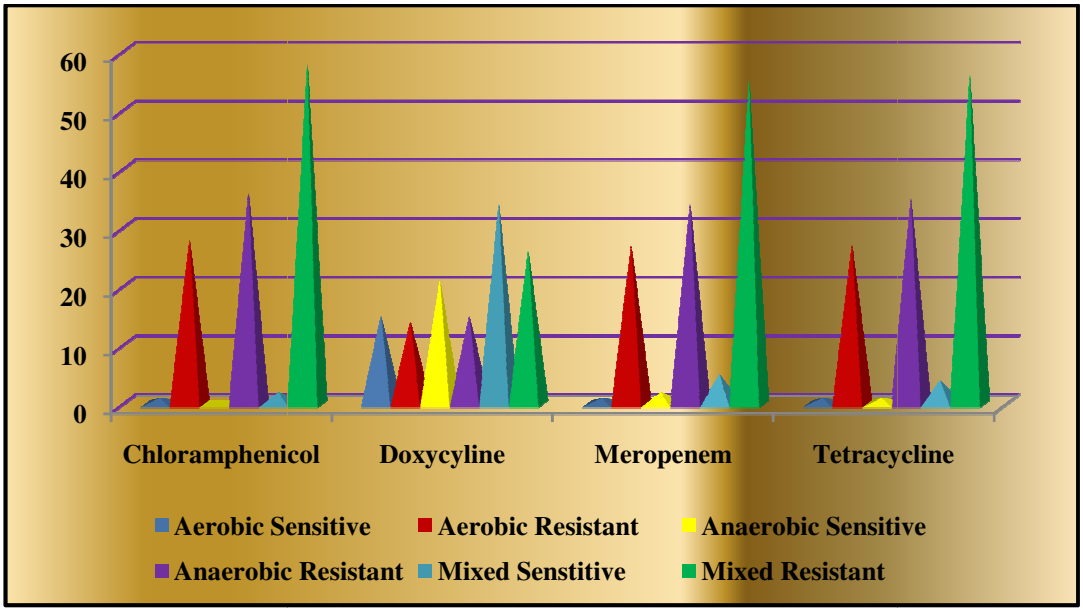
Graph 14: Number of cases sensitive and resistant to Cephalosporin group



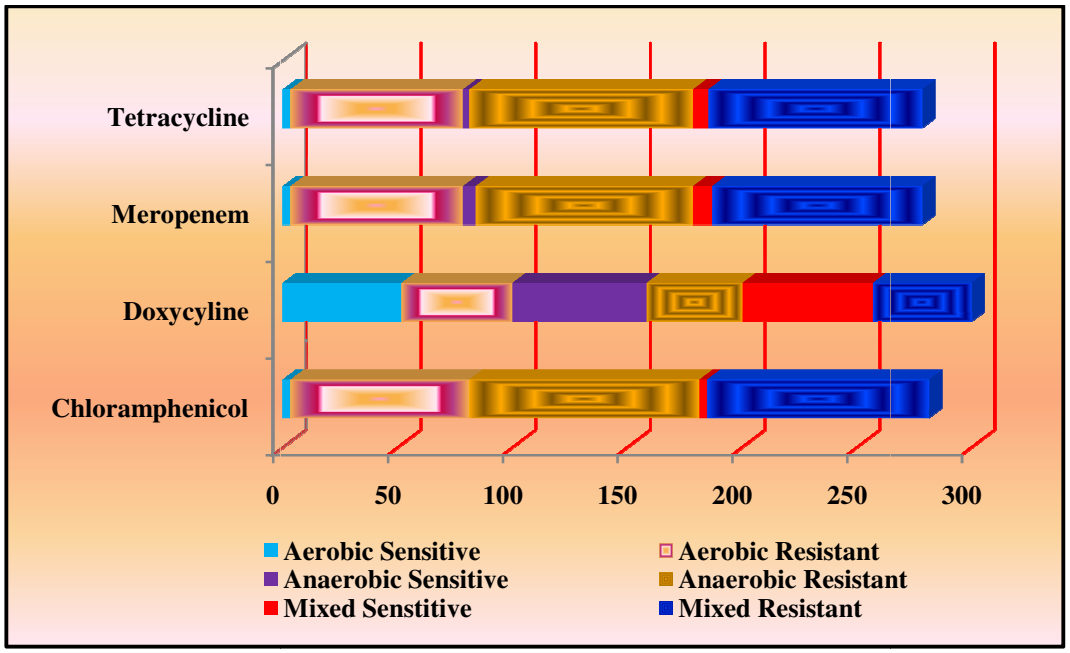
Graph 15: Percentage of cases sensitive and resistant to Cephalosporin group



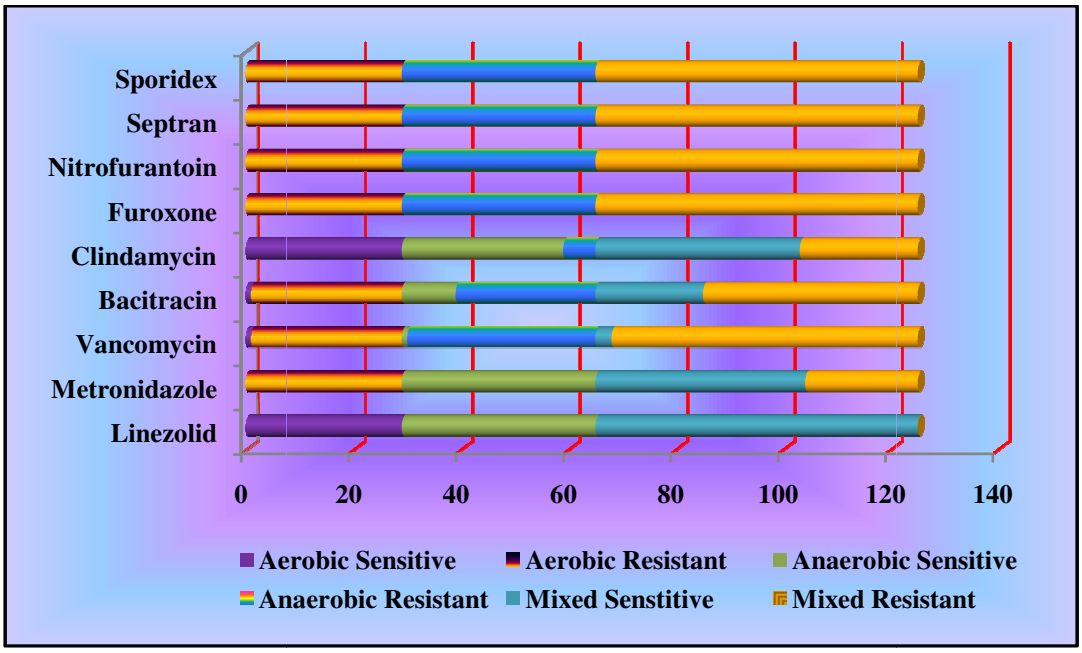
Graph 16: Number of cases sensitive and resistant to Broad spectrum antibiotics



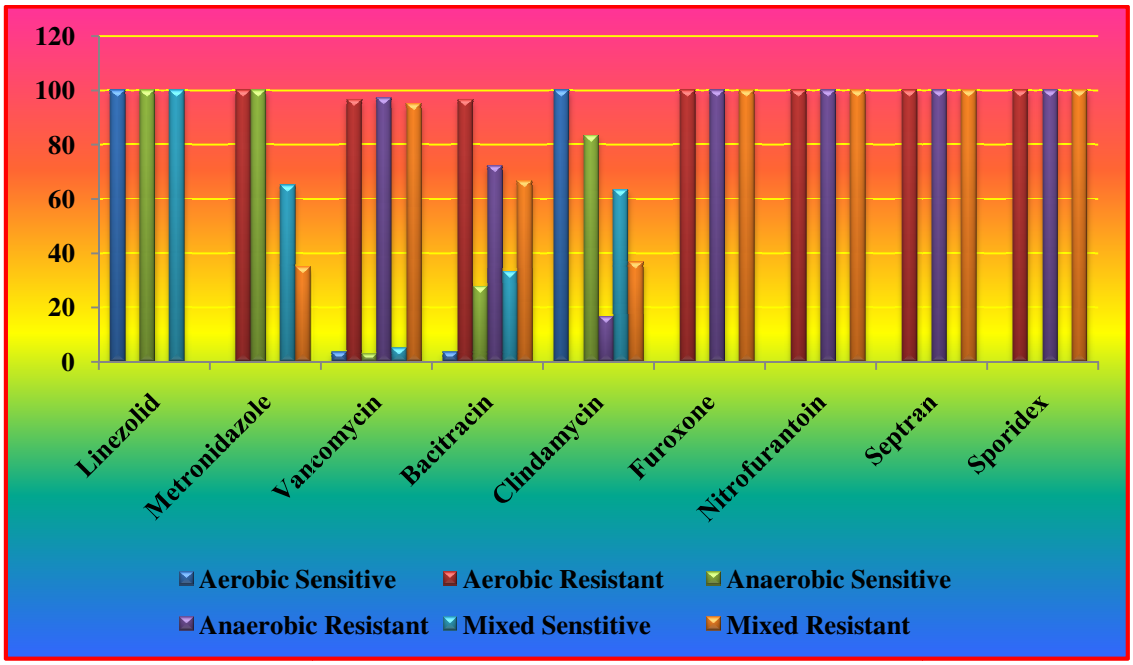
Graph 17: Percentage of cases sensitive and resistant to Broad spectrum antibiotics



Graph 18: Number of cases sensitive and resistant to miscellaneous group of drugs



Graph 19: Percentage of cases sensitive and resistant to miscellaneous group of drugs



Figures

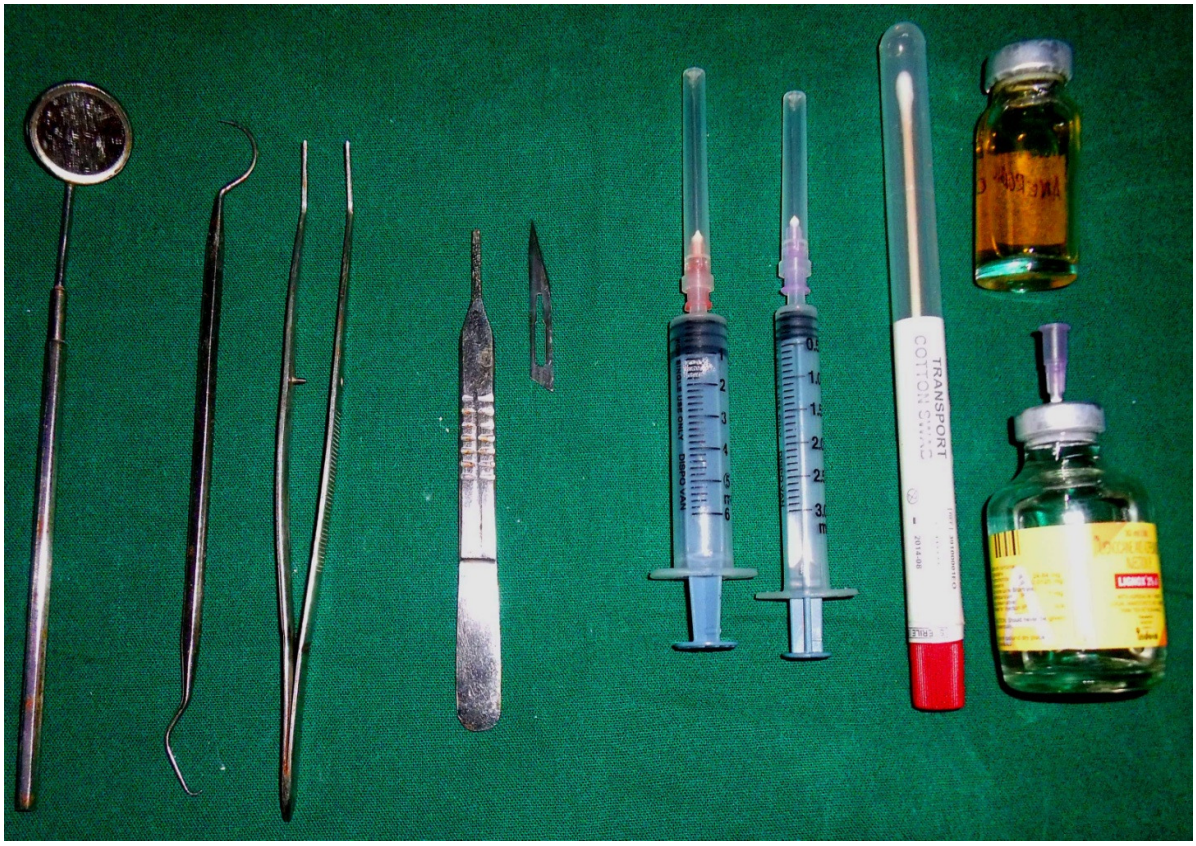


Fig 1:-Armamentarium

Fig 2:- Transport medium (Brain heart infusion broth and Transport cotton swab)

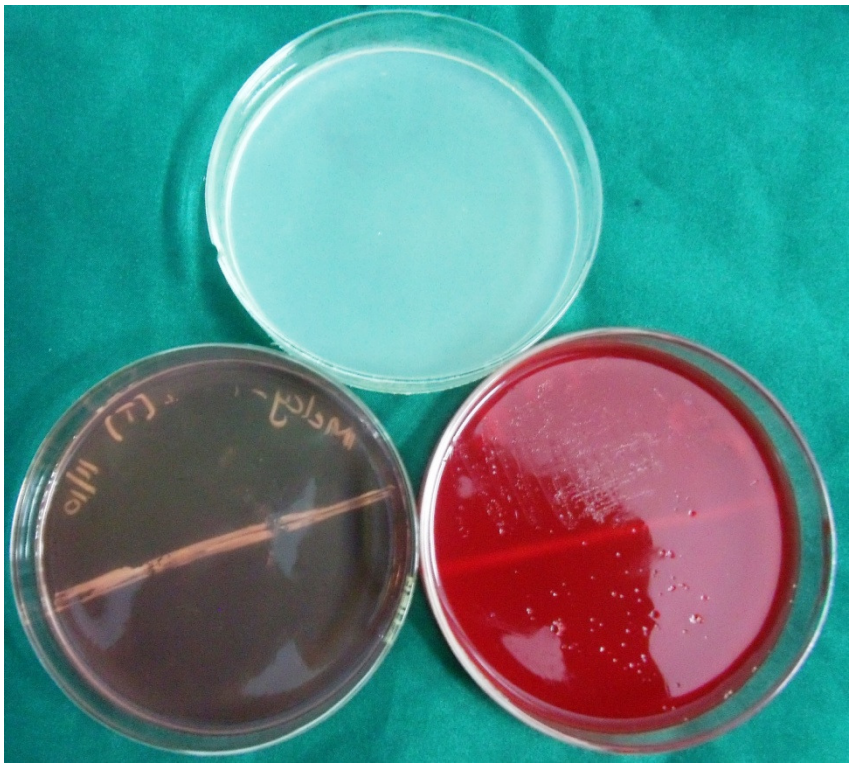


Fig 3:- Nutrient Agar plate, Maconkey's agar plate, Blood agar plate

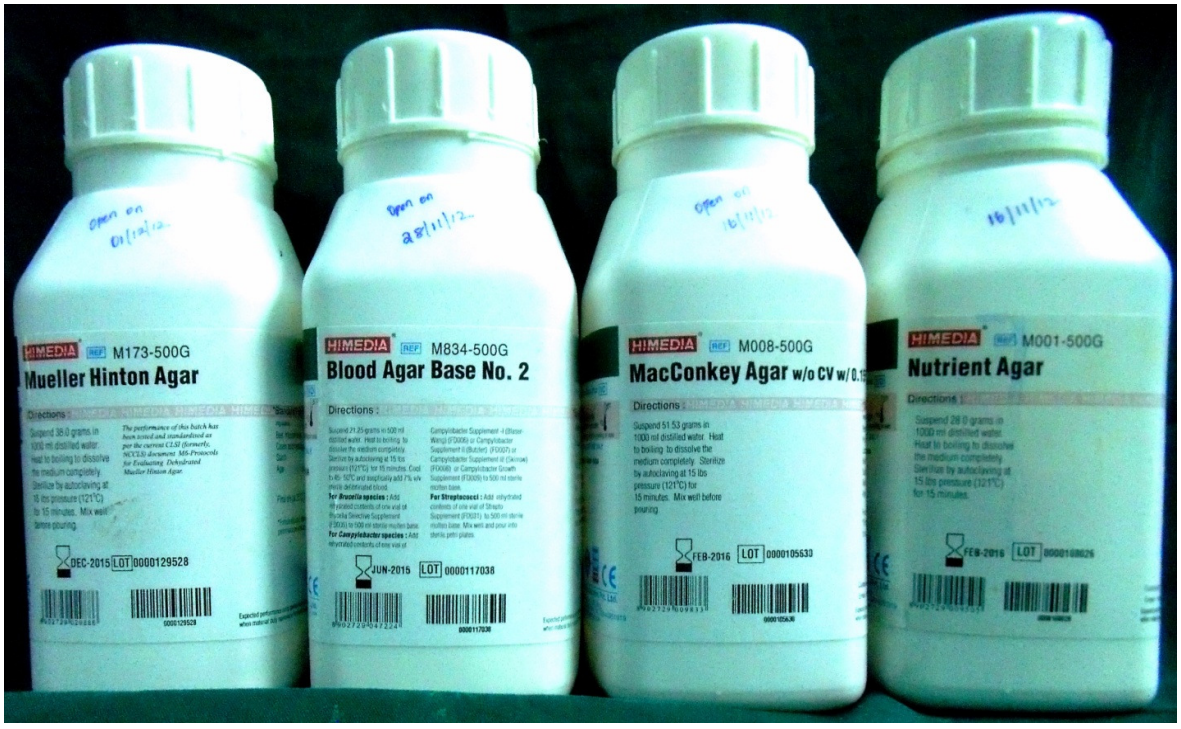


Fig 4:- Mueller Hinton Agar, Blood Agar, MacConkey Agar, and Nutrient Agar,

Fig 5:-Flame sterile wire loop

Fig 6:- Inoculation into the blood agar plate

Fig 7:- Streaking

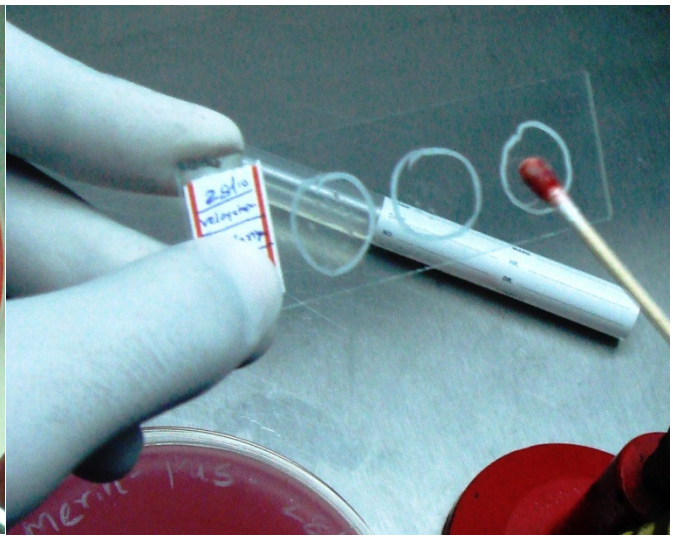


Fig 8:- Gram staining

Fig 9:- Incubator

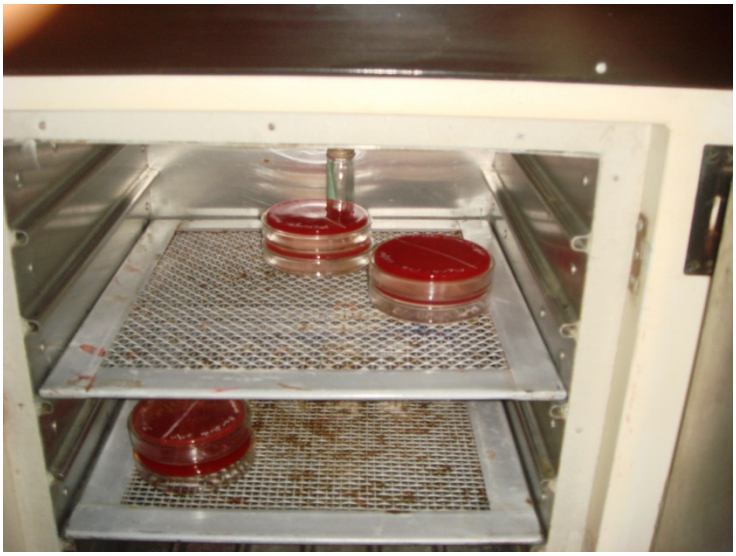


Fig 10:- Incubator with media plates and transport media

Fig 11:- Anaerobic Jar

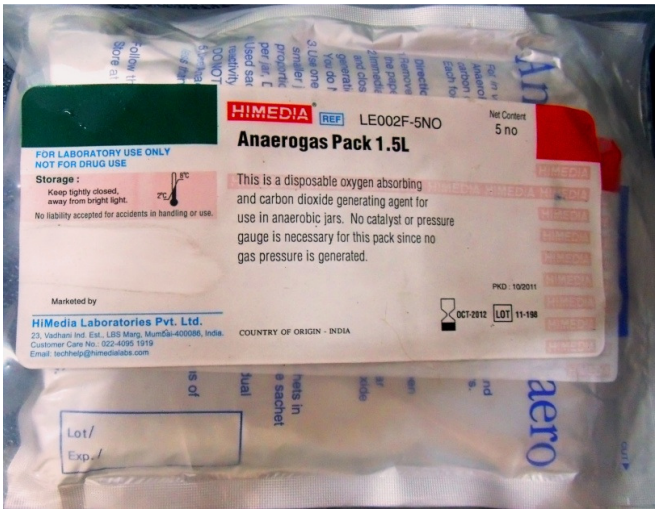


Fig 12:- Gas Pack

Fig 13:- Gas Pack with indicator tablet

Fig 14:- Anaerobic jar with media plates

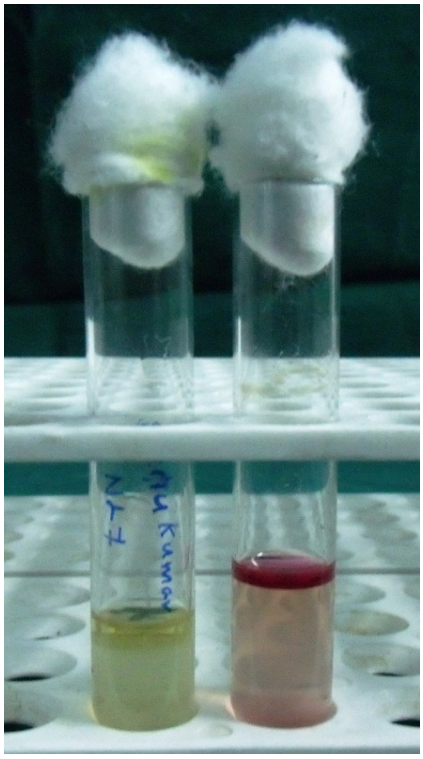


Fig 15:- Indole test- Negative and Positive

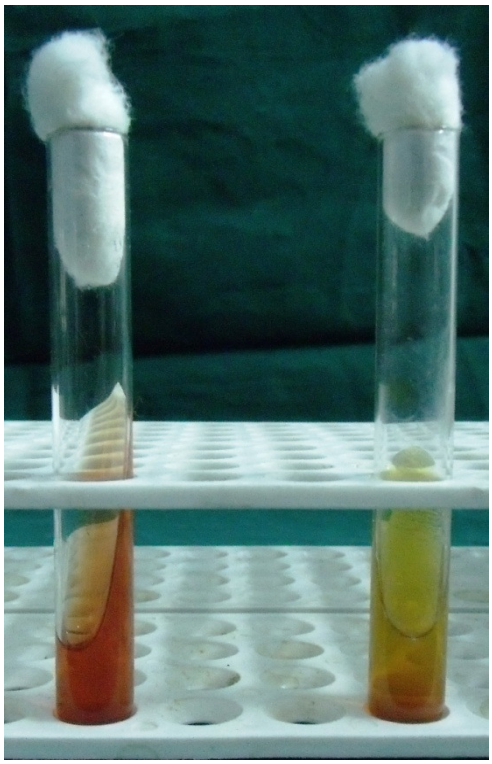


Fig 16:- Triple sugar iron test- Negative and Positive

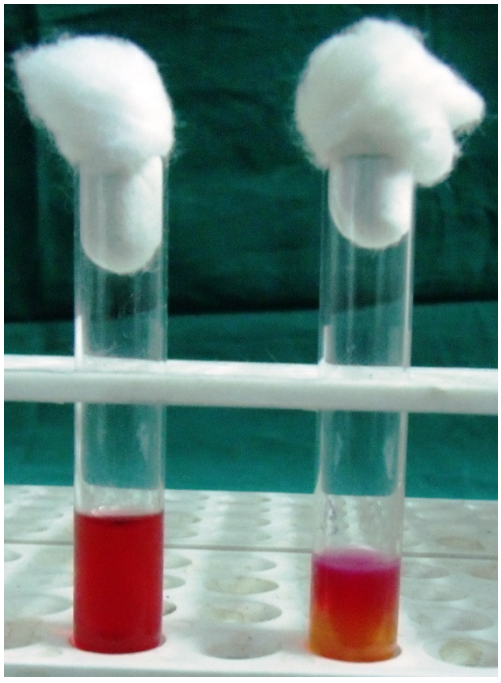


Fig 17:- Mannitol motility test- Negative and Positive.

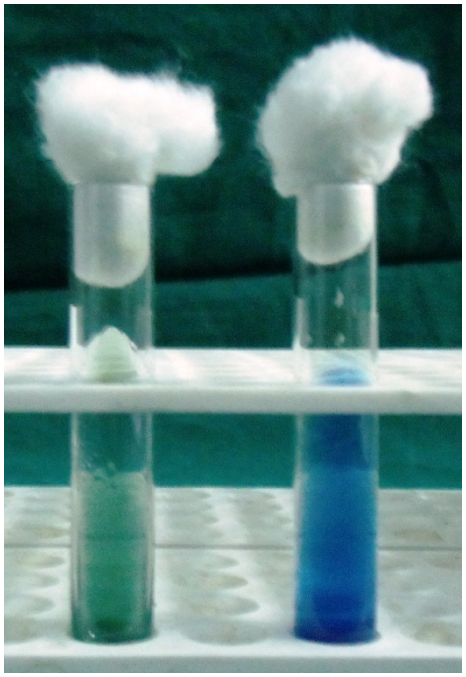


Fig 18:- Citrate test- Negative and Positive

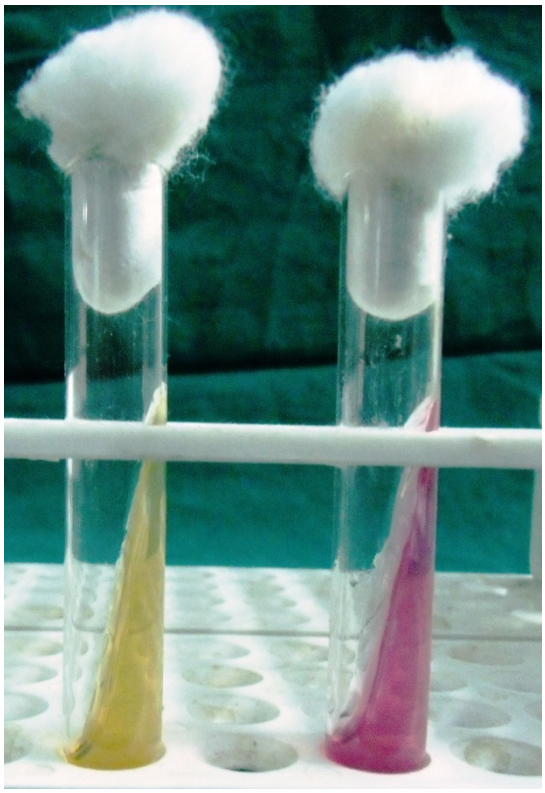


Fig 19:- Urase test- Negative and Positive

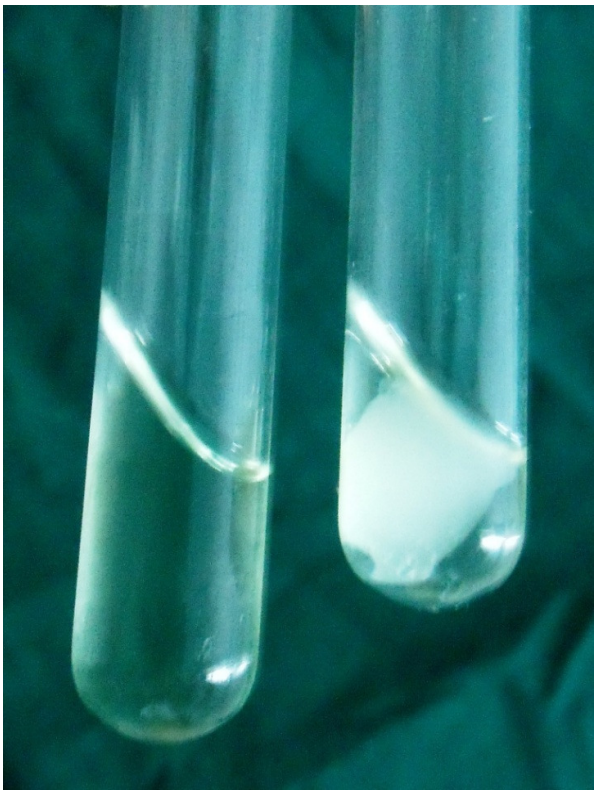


Fig 20:- Coagulase test- Negative and Positive

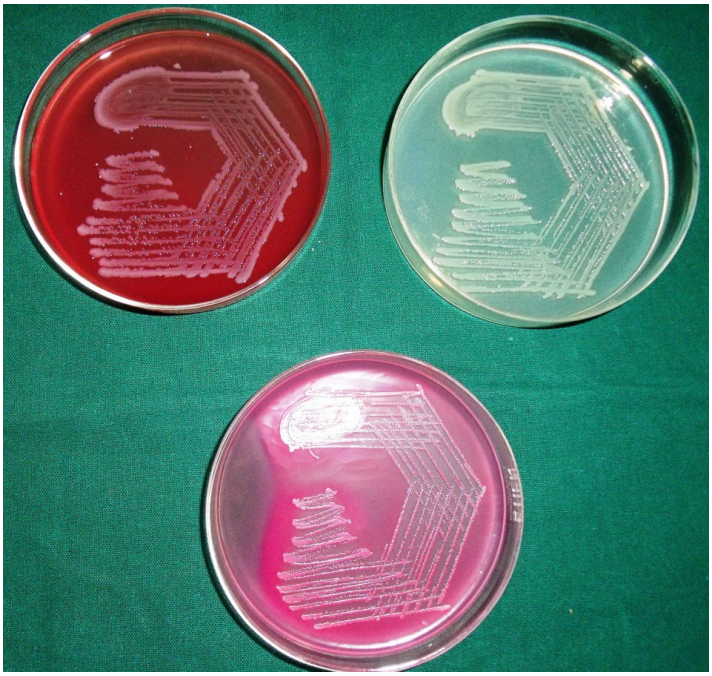


Fig 21:- E.Coli

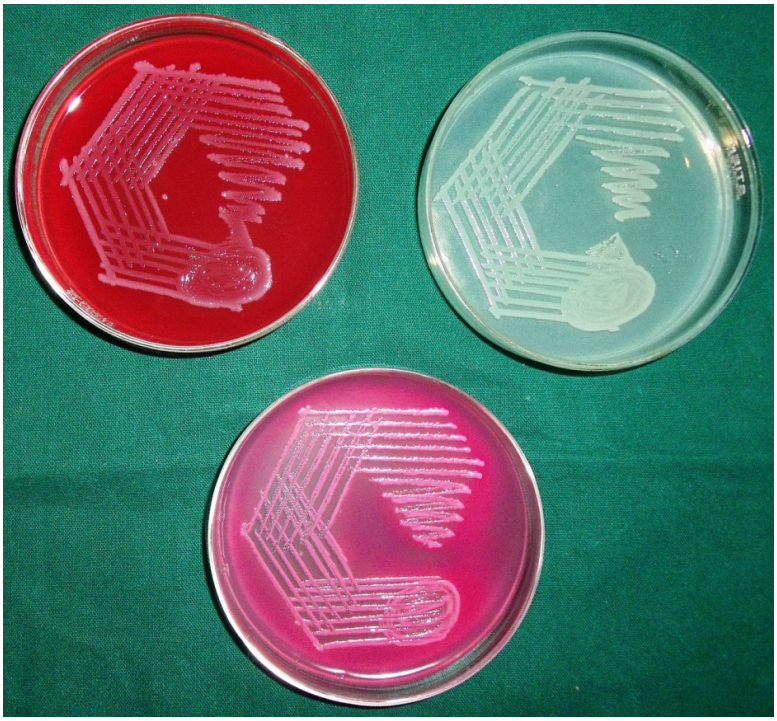


Fig 22:- Klebsiella Pneumonia

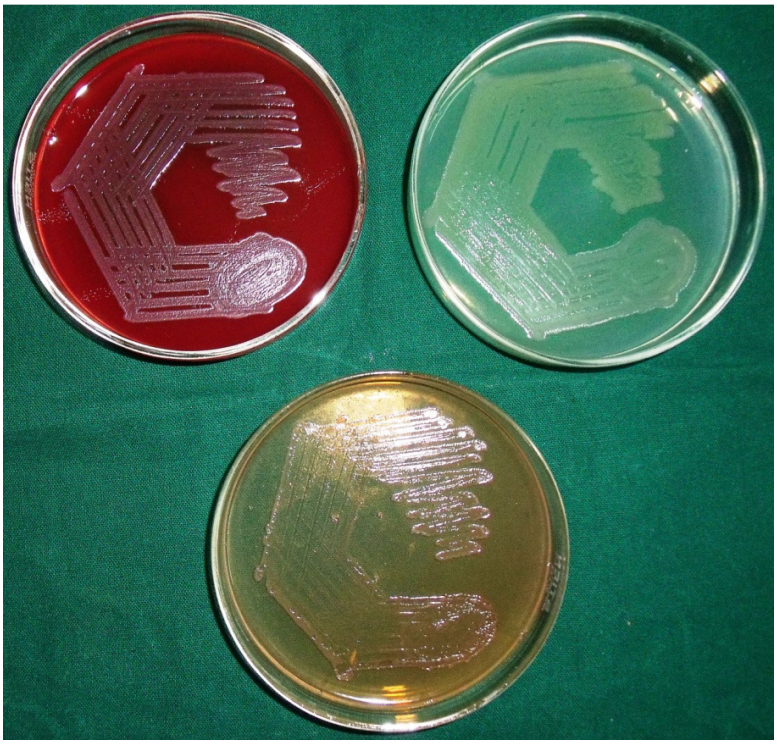


Fig 23:- Pseudomonas Aerogenosa

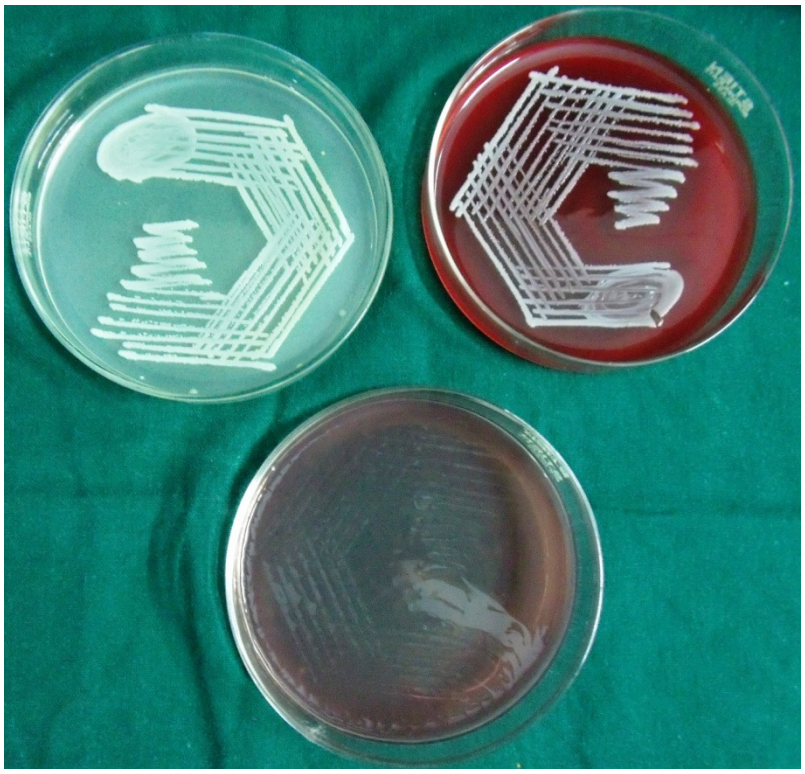


Fig 24:- Staphylococcus aureus

Fig 25:- Streptococcus viridians

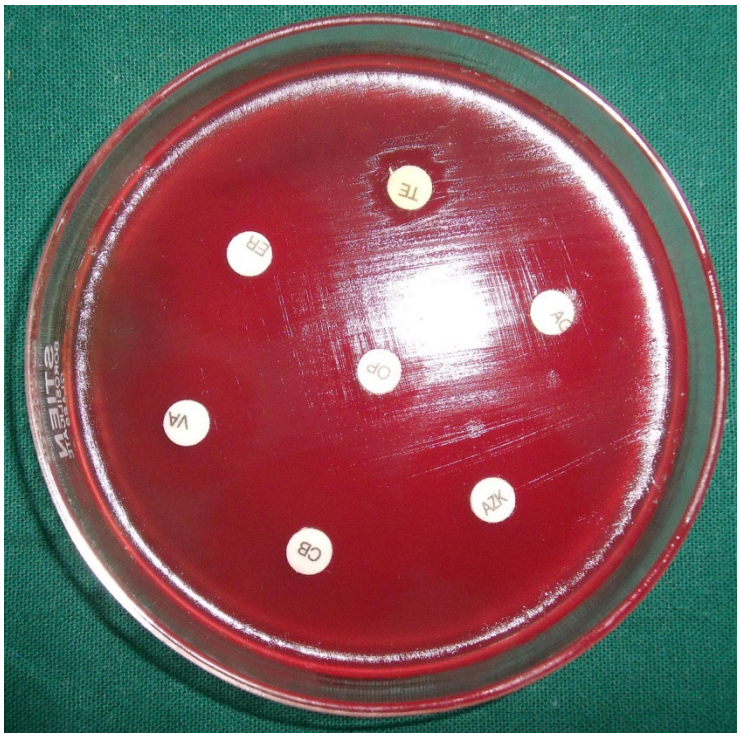


Fig 26:- Streptococcus Viridans- Optochin confirmation test

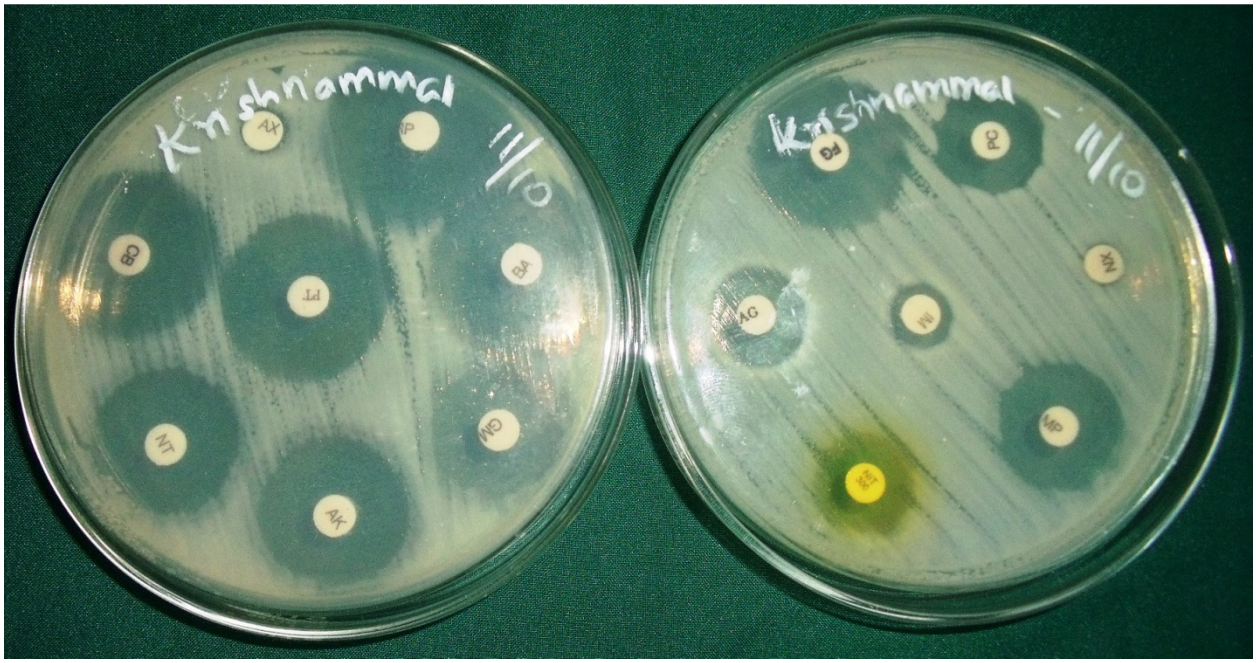


Fig 27:- Mueller Hinton Agar plate with Antimicrobial disc showing antibiotic sensitivity

Case no-1

Fig 28:-Vestibular space infection

Fig 29:- Administering local anesthesia

Fig 30:- Intraoral incision

Fig 31:- collection of pus sample with swab

Case no-2

Fig 32:-Vestibular space infection

Fig 33:- Administering local anesthesia

Fig 34:- Aspiration of pus sample

Fig 35:- Pus sample transferred into the transport media

Bibliography

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Appendix

Case History Performa

Name :

Age/Sex :

O.P.No :

Address :

Chief complaint :

History of Present Illness (HOPI):

Past Medical History:

Past dental History:

History of drug allergy:

General Examination:

Extra/Intra oral Examination:

Provisional Diagnosis:

Treatment done: